THESIS

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

SESSION 2022-2025

"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS"

DR SWARNIM BAIDYA

UNDER SUPERVISION OF

DR. SWATI SINGH

DEPARTMENT OF PATHOLOGY

SANTOSH MEDICAL COLLEGE & HOSPITAL

GHAZIABAD, NCR DELHI

SANTOSH DEEMED TO BE UNIVERSITY

NO. 1, SANTOSH NAGAR,

GHAZIABAD-201009, NCR DELHI, INDIA

COPYRIGHT CERTIFICATE-DECLARATION BY THE CANDIDATE

I hereby declare that Santosh Deemed to be University, Ghaziabad, (U.P.) shall have the right to preserve, use and disseminate this thesis titled **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS"** in print or electronic format, for academic or research purpose.

Signature of Candidate:

Date :

Place :

CERTIFICATE BY THE GUIDE

This is to certify that the thesis titled **"CLINICO-HISTOPATHO LOGICAL STUDY OF PIGMENTED SKIN LESIONS"** is a bonafide genuine research work carried out by **Dr. Swarnim Baidya** in partial fulfilment of the requirements for award of the degree of M.D. in the speciality of Pathology, under my guidance and supervision and is completed to my full satisfaction.

Guide

Dr. Swati Singh
Professor
Department of Pathology
Santosh Medical College & Hospital
Ghaziabad NCR, India

CERTIFICATE BY THE CO-GUIDE

This is to certify that the thesis titled **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS"** is a bonafide genuine research work carried out by **Dr. Swarnim Baidya** in partial fulfilment of the requirements for award of the degree of M.D. in the speciality of Pathology.

Co-Guide

Dr. Richa Sharma

Assistant Professor

Department of Pathology

Santosh Medical College & Hospital

Ghaziabad NCR, India

CERTIFICATE BY THE HEAD OF THE DEPARTMENT

This is to certify that the thesis titled **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS"** is a bonafide research work carried out by **Dr. Swarnim Baidya** in partial fulfilment of the requirements for award of the degree of M.D. Pathology will be based on bonafide work carried out by her.

PROF. (Dr.) Adreena Mittal
M.B.B.S., M.D.
Professor & Head
Department of Pathology
Santosh Medical College & Hospital
Ghaziabad, NCR, India

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

ENDORSEMENT BY THE DEAN OF THE COLLEGE

This is to certify that the thesis titled **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS"** is a bonafide research work carried out by **Dr. Swarnim Baidya** under the esteemed guidance **of Dr. Swati Singh**, **Professor, Department of Pathology, Santosh Medical colleg**e and is presented before the ethical committee **of Santosh University, Ghaziabad** for requisite approval.

Dr Alka Agrawal

Dean

Faculty of Medical Sciences

Santosh Medical College & Hospital

Santosh Deemed to be University

Ghaziabad, NCR, India

ACKNOWLEDGMENT

At the outset, I thank The Lord Almighty for giving me the strength to perform all my duties.

I would express my humble gratitude and sincere thanks to my guide, **Dr. Swati Singh,** MD, Professor, Department of Pathology, Santosh Medical College and Hospital, Ghaziabad, for her encouragement and kind support. It is my privilege to have worked under her valuable guidance, whose supervision and encouragement have immensely helped in the successful accomplishment of this study.

I would express my humble gratitude and sincere thanks to my Head of the department, **Dr. Adreena Mittal,** MD, Professor and Head, Department of Pathology, Santosh Medical College and Hospital, Ghaziabad, for her encouragement and kind support.

My sincere thanks and heartfelt gratitude to my co-guide, **Dr. Richa Sharma**, MD, Assistant Professor, Department of Pathology, for her continuous support and suggestions which led to the successful completion of this study.

I am grateful to **Dr. Alka Agrawal,** Dean, Santosh Medical College and Hospital, Ghaziabad, for permitting me to utilize the college and hospital facilities for my dissertation.

I am grateful to my colleagues, and my juniors who always lent their helping hands.

My sincere thanks to all the technical staff of the Department of Pathology, Santosh Medical College and Hospital, Ghaziabad, for their help.

My deepest thanks go to my caring parents, **Dr. Ashok Kumar Baidya** and **Mrs. Amrita Baidya**, my supportive in-laws, **Mr. Awadhesh Prasad** and **Mrs. Sushila Kumari**, my loving husband, **Dr. Ashutosh Kumar,** and my supportive sister, **Dr Shubhra Baidya,** for their unwavering support and help throughout this journey.

I express my sincere thanks and gratitude to all the patients who form the backbone of this study without whom this would not have been possible.

With sincere thanks,

Dr Swarnim Baidya

LIST OF ABBREBIATIONS

1. **HCP** - Histopathological and Clinical Pathology
2. **PSL** - Pigmented Skin Lesions
3. **HCL** - Histopathological and Clinical Correlation
4. **HS** - Histopathology Study
5. **PCL** - Pigmented Lesion Correlation
6. **HC** - Histopathological-Clinical
7. **SLD** - Skin Lesion Diagnosis
8. **HCR** - Histopathological-Clinical Relationship
9. **LP** - Lesion Pathology
10. **TCC** - Tertiary Care Center
11. **HD** - Histopathological Diagnosis
12. **PCF** - Pigmented Lesion Clinical Features
13. **SR** - Skin Lesion Review
14. **HPCF** - Histopathological and Clinical Features
15. **LSC** - Lesion Skin Characteristics

INDEX

S. No.	Title	Page Number
1	Introduction	1-5
2	Aim and Objective	6
3	Review of Literature	7-23
4	Material and Method	24-26
5	Results	27-42
6	Discussion	43-55
7	Conclusion	56
8	Limitations	57
9	Summary	58-59
10	References	60-72
11	Master chart	73-78
12	Proforma	79-80
13	Consent form- English	81
14	Consent form- Hindi	82
15	Ethical Clearance	83
16	Dissertation Workshop Certificate	84
17	Plagiarism Certificate	85

Introduction

INTRODUCTION

The skin stands as the body's largest organ, constituting approximately 15% of an adult human's total body weight.[1] The significance of precise diagnosis cannot be overstated, particularly considering the diverse aetiologies underlying various diseases. Accurate diagnosis is paramount for the effective treatment of hyperpigmented lesions. In this comprehensive analysis, we delve into the pivotal role of histology in diagnosing hyperpigmented disorders. We explore how histological analysis not only aids in diagnosis but also paves the way for targeted and more efficacious therapeutic interventions. Our focus centers on elucidating the most prevalent hyperpigmented disorders observed among Indian patients.[2] The skin, being the largest organ in the human body, plays a multifaceted role in maintaining homeostasis and safeguarding the body from external threats. Its complex structure comprises several layers, each contributing uniquely to its functions. Notably, the outermost layer, the epidermis, serves as a protective barrier, shielding the body from harmful pathogens and environmental stressors. Beneath the epidermis lies the dermis, which houses essential structures such as blood vessels, hair follicles, and sweat glands, crucial for thermoregulation and nutrient exchange.

Hyperpigmentation, characterized by the excessive production of melanin, presents a common dermatological concern, affecting individuals of all ages and ethnicities. While often benign, hyperpigmented disorders can significantly impact one's quality of life and self-esteem. Moreover, they may signify underlying systemic conditions necessitating thorough evaluation and management.

Histological examination emerges as a pivotal tool in the accurate diagnosis of hyperpigmented disorders. By scrutinizing tissue samples under a microscope, dermatopathologists can discern subtle architectural changes and cellular abnormalities indicative of specific dermatoses. This microscopic insight not only

confirms the diagnosis but also provides valuable prognostic information, guiding clinicians in tailoring treatment strategies to individual patients.

In the context of Indian patients, certain hyperpigmented disorders exhibit unique clinical and histological features, necessitating a nuanced approach to diagnosis and management. Conditions such as melasma, post-inflammatory hyperpigmentation, and lichen planus pigmentosus are prevalent among this demographic and often pose diagnostic challenges due to overlapping clinical presentations.

Melasma, characterized by symmetric hyperpigmented patches typically affecting sun-exposed areas, represents a prime example of a hyperpigmented disorder commonly encountered in Indian patients. Histologically, melasma manifests as increased melanin deposition within the epidermis and dermis, accompanied by subtle alterations in keratinocyte morphology. Distinguishing melasma from other pigmentary disorders, such as post-inflammatory hyperpigmentation or drug-induced pigmentation, requires meticulous histopathological evaluation and correlation with clinical findings.

Similarly, post-inflammatory hyperpigmentation, arising as a sequelae to various inflammatory skin conditions or trauma, presents histological features reminiscent of its inciting factors. Histopathological examination typically reveals increased melanin production within basal keratinocytes and melanophages scattered throughout the dermis. The presence of dermal inflammation and architectural distortion further aids in confirming the diagnosis.

Lichen planus pigmentosus, a distinct variant of lichen planus predominantly affecting individuals with Fitzpatrick skin types IV-VI, poses diagnostic dilemmas due to its overlapping clinical and histological features with other hyperpigmented disorders. Histopathological examination of lesional skin reveals a band-like lymphocytic infiltrate at the dermo-epidermal junction accompanied

by pigment incontinence and melanophages within the papillary dermis, thus corroborating the clinical diagnosis.

In conclusion, histology serves as an indispensable tool in unraveling the intricate pathogenesis of hyperpigmented disorders, offering invaluable insights into disease mechanisms and guiding personalized therapeutic interventions. By leveraging histological findings in conjunction with clinical data, clinicians can optimize patient care, ensuring accurate diagnosis and tailored management strategies tailored to the unique needs of Indian patients and beyond.

Pigmentary issues are a common reason people see a dermatologist. These problems happen when there are issues with the pigment cells in the skin. This can be because these cells didn't move to the right places in the skin when the body was developing before birth. Another reason can be that there's a problem with how these cells pass on their pigment to other skin cells, or there could be a change in how pigment is made.[2]

Even though having more pigment in the skin can protect it from sunlight, it's still possible to have skin damage from the sun that shows up as pigmentary issues.

Hyperpigmentation presents not just as a mere cosmetic inconvenience but also as a potential indicator of underlying organ irregularities. When patients present themselves with skin lesions, often indicative of melanocyte activity, accurate diagnosis becomes imperative for appropriate treatment. However, diagnosing skin diseases poses a challenge due to the overlapping clinical information and appearance of lesions. In many cases, confirming the diagnosis without histopathologic examination of skin biopsies proves difficult. Histologic diagnosis, especially in inflammatory and various skin diseases, necessitates clinicopathologic correlation, considering the evolving nature of skin lesions throughout the disease progression. Thus, histopathologic examination serves as both a confirmative and complementary aspect of diagnosis. Leveraging

histology-based insights, treatment and management strategies can be refined to establish more precise protocols for addressing hyperpigmented skin lesions.[3]

Expanding on this, it's crucial to underscore the multifaceted nature of hyperpigmentation's impact. Beyond its surface-level manifestation, it often hints at deeper physiological imbalances within the body. Consequently, when individuals seek medical attention for hyperpigmented skin, physicians confront the challenge of accurately diagnosing the underlying cause amidst the myriad of potential skin conditions. The complexity is further compounded by the fact that clinical presentations and visual cues may overlap across different skin diseases.

Given this diagnostic puzzle, histopathologic examination emerges as an indispensable tool in unraveling the intricacies of hyperpigmentation-related disorders. Through the microscopic analysis of skin biopsies, clinicians gain invaluable insights into the histological characteristics and pathological mechanisms underlying these conditions. This histological perspective not only confirms diagnoses but also sheds light on the evolving nature of skin lesions as diseases progress.

Moreover, histology doesn't merely serve as a standalone diagnostic modality but rather complements clinical observations, forming a synergistic approach to accurate diagnosis and treatment planning. By integrating histopathologic findings into the diagnostic framework, healthcare professionals can tailor treatment protocols to address the specific histological features driving hyperpigmented skin lesions. This precision-guided approach holds immense promise in optimizing therapeutic outcomes and enhancing patient care.

In essence, while hyperpigmentation may superficially manifest as a cosmetic concern, its significance extends far beyond skin deep. By leveraging histopathologic insights, clinicians can navigate the complexities of diagnosis and

treatment, ultimately empowering individuals to overcome the challenges posed by hyperpigmentation-related disorders.

Pigmentary disorders represent a prevalent dermatological concern that transcends geographical boundaries, manifesting in various forms across diverse populations worldwide. These acquired pigmentary irregularities, while ubiquitous, exhibit classic morphologies that have garnered significant attention within the global dermatological community. Comprehensive analyses and discussions of these distinctive lesions have been extensively documented in dermatology literature on a global scale, reflecting the collective efforts to understand and effectively address these challenging conditions. Moreover, emphasis has been placed on developing treatment strategies tailored to manage the complexities associated with these pigmentary disorders, reflecting the ongoing commitment to enhancing patient care and outcomes in dermatological practice.

Aims

&

Objectives

AIM & OBJECTIVE

AIM - The proposed study entitled as:

"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS" was conducted to establish a relationship between histopathological and clinical features of pigmented skin lesions.

OBJECTIVE--

- To correlate the histopathological and clinical findings of pigmented skin lesions.
- To study the role of histopathology in diagnosis of pigmented skin lesions.

Review Of Literature

REVIEW OF LITERATURE

Patel et al. conducted a study in 2013[4] aiming to establish a clinicopathological correlation of acquired hyperpigmented disorders. The study emphasized the significance of accurate diagnosis due to the varying etiologies underlying these disorders, particularly prevalent among Asians and Indians. The authors highlighted the utility of histopathological evaluation in diagnosing hyperpigmented disorders, leading to more targeted and effective therapy. Various common and uncommon conditions were explored, including facial melanoses, lichen planus pigmentosus, discoid lupus erythematosus, and others.

Rajput et al (2017)[5] conducted a clinico-pathological study of non-neoplastic skin disorders to analyze the correlation between clinical diagnosis and histopathological examination. The study involved examining skin biopsies from patients with non-neoplastic skin lesions, revealing a variety of clinicopathological patterns. The authors concluded that skin biopsy is a valuable investigative procedure for confirming clinical diagnoses, particularly in cases of infectious diseases.

Walker et al. (2017)[6] conducted a cross-sectional study to describe histopathological features in morphea (localized scleroderma) and their clinical correlates. Their findings suggested associations between microanatomical location, degree of sclerosis, and inflammation seen on histologic samples with patient-reported symptoms and physician-based measures of severity. The study emphasized the importance of histopathological examination in identifying patients requiring additional monitoring and treatment for morphea.

Samanta et al.(2018)[7] conducted a histopathological study of skin tumors to address the diagnostic challenges associated with skin tumors. The study analyzed 52 cases of skin tumors and emphasized the importance of light microscopic examination as the standard technique for diagnosis. Benign and

malignant skin tumors were characterized, with intradermal nevus being the most common benign tumor and squamous cell carcinoma being the most common malignant tumor.

Goswami and Sharma et al.,(2020)[9] reviewed the use of herbal extracts for treating skin hyperpigmentation disorders. They highlighted the increasing preference for herbal products over synthetic ones due to their mild nature and low toxicity. The review discussed the potential benefits of herbal extracts in improving skin conditions, emphasizing their efficacy and safety compared to synthetic formulations.

Day et al.[10] described clinicopathologic diagnostic criteria for vulvar lichen planus (LP) in a study published in 2020. The study aimed to provide comprehensive criteria for diagnosing erosive, classic, and hypertrophic types of vulvar LP. Clinicopathological correlation was emphasized as crucial for accurate diagnosis, considering the overlaps with other dermatological conditions. The study recommended a low threshold for biopsy and highlighted the utility of histopathological criteria in reducing nondiagnostic biopsy rates.

Ravindran et al. (2020)[11] conducted a study to establish histopathological and clinical correlations of hyperpigmented skin lesions. Their research included histopathological examination of punch skin biopsy lesions from 100 patients with hyperpigmented lesions. The study identified various hyperpigmented skin conditions, with classical lichen planus and its variants being the most common.

Gangadhar et al. (2021)[12] conducted a prospective cross-sectional study to establish correlations between clinical, dermoscopic, and histopathological findings in follicular keratotic diseases. The study aimed to identify dermoscopic criteria for diagnosing primary follicular keratotic diseases. Their findings revealed significant associations between clinical, dermoscopic, and

histopathological features, highlighting the utility of dermoscopy as a non-invasive diagnostic tool for differentiating follicular keratotic diseases.

Skin lesions:

Skin lesions with coloration can be described as having shades of brown, black, or blue. Sometimes, these lesions might appear similar to brown or black spots even when they're not. For instance, there are types of lesions, known as vascular lesions, that might seem black when you look at them without any special tools. However, when examined under a dermatoscope, a tool used by dermatologists to get a closer look at the skin, these vascular lesions show their true colors, which can range from red and purple to blue.[14-18] This tool helps in distinguishing these lesions from those that are genuinely brown or black, providing a clearer understanding of their nature and aiding in accurate diagnosis.

The colors seen in pigmented skin lesions have different sources. Firstly, melanin, a natural pigment found in the skin, is a primary reason for the color variation in these lesions.[19] This pigment is responsible for the dark hues observed. Secondly, blood can influence the color of skin lesions, giving them a redder or even purplish appearance, depending on the amount and state of the blood present within the lesion. Lastly, external factors can also contribute to the color of skin lesions. For example, tattoos, which involve the insertion of pigment into the skin, can affect the appearance and color of these areas. This variety of sources for pigmentation explains why skin lesions can appear in so many different colors and shades. Skin color changes or spots often involve melanocytes, the cells responsible for skin pigment.[20-25] But, it's not just melanocytes that can cause pigmented skin changes. People, especially those with darker skin tones, might also have pigmented changes from non-melanocytic sources. These include changes related to the cells that make up the skin's structure (keratinocytes), blood vessels, or even reactions to various factors.[26]

When we talk about melanocytic lesions, these are usually related to the natural or benign growths known as moles. Moles can vary widely in appearance and type, including those known as blue naevi. On the other hand, there's melanoma, which is a serious form of skin cancer arising from melanocytes. It's important to understand the difference between these conditions, as they can have different implications for health and require different approaches to treatment.

Melanocytic naevus

Congenital naevus

Blue naevus

Melanoma

Skin conditions involving the keratinocytes, which are the predominant type of cells found in the epidermis (the outer layer of the skin), can vary widely in their appearance and seriousness. Among these conditions, we have several notable types:

1. Seborrhoeic Keratosis: This is a common, non-cancerous growth that appears on the skin. It's often brown, black, or light tan and has a waxy, scaly, slightly elevated appearance. These lesions can appear anywhere on the body except the palms and soles and are more common in older adults.

2. Lentigo: Lentigines, or what we commonly refer to as freckles, come in various forms. Solar lentigo, for instance, is a type of freckle that results from sun exposure. These spots are usually small, flat, and brown.

3. Epidermal Naevus: This term encompasses a group of non-cancerous skin growths that include Becker's naevus. These growths result from an overgrowth of cells in the epidermis and can vary in size, shape, and color.

4. Pigmented Basal Cell Carcinoma: Basal cell carcinoma is a type of skin cancer that can appear as a pigmented lesion, making it look somewhat similar to a mole. It's the most common type of skin cancer but tends to grow slowly and is usually curable.

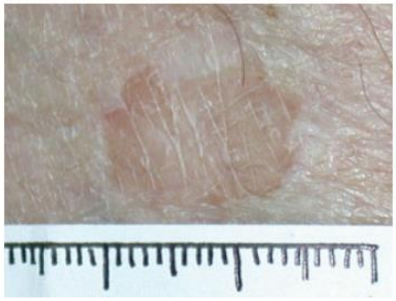
5. Pigmented Actinic Keratosis and Intraepidermal Carcinoma: Actinic keratosis is a rough, scaly patch on the skin that develops from years of exposure to the sun. It's considered a precancerous condition that can sometimes progress to squamous cell carcinoma. Intraepidermal carcinoma, also known as Bowen's disease, is a form of squamous cell carcinoma that's confined to the outer layer of the skin.

6. Pigmented Squamous Intraepithelial Lesions: These lesions can occur on the vulva, penis, and anus and represent early forms of skin cancer that haven't yet invaded deeper tissues.

7. Pigmented Invasive Squamous Cell Carcinoma: This is a more advanced form of squamous cell carcinoma that has penetrated beyond the outer layer of the skin and can spread to other parts of the body.

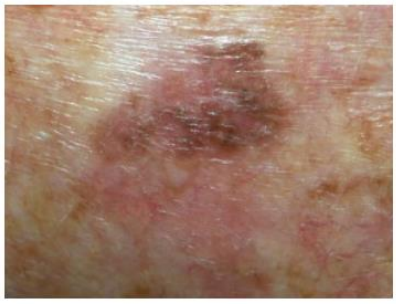
8. Pigmented Keratinocytic Lesions: This is a general term that can refer to any of the aforementioned conditions when they present with pigmentation.

Seborrhoeic keratosis



Solar lentigo

Basal cell carcinoma



Intraepidermal carcinoma

Vascular lesions are problems related to the blood vessels in the body. These can occur for various reasons and manifest in different ways.[27] For example, if someone experiences a minor injury, like getting a small piece of wood under their nail, it might cause a splinter haemorrhage, which appears as a dark line under the nail. Similarly, bruising can lead to purpura, a condition where purple spots appear on the skin due to bleeding underneath. This can also be the result of capillaritis, where inflammation affects the small blood vessels, or vasculitis, where blood vessels become inflamed more broadly.[28]

There are also specific types of growths associated with vascular lesions. A cherry angioma is a small, round, and red growth that can pop up anywhere on the body. It's quite common and usually not a cause for concern. Angiokeratoma is another type, presenting as dark spots on the skin that can feel rough or bumpy. While these are typically harmless, they can sometimes bleed if injured.

More serious vascular lesions include angiosarcoma and Kaposi sarcoma. Angiosarcoma is a rare cancer that affects the lining of the blood vessels and can appear anywhere in the body, often as a skin lesion that looks bruised or

purplish.[21] Kaposi sarcoma is another type of cancer, more commonly associated with a weakened immune system, such as in people with HIV/AIDS. It also appears as dark lesions on the skin or internally.

Splinter haemorrhage

Cherry angioma

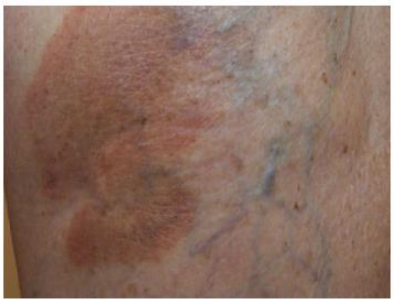
Kaposi sarcoma

Angiokeratoma

Reactive pigmentation refers to changes in skin color that occur in response to various conditions.[29-32] This can include a range of skin issues, from dermatofibroma, which are non-cancerous growths that can develop anywhere on the skin, to dermatofibrosarcoma protuberans, a more serious type of skin cancer that grows slowly but can spread to other parts of the body if not treated. Another common cause of reactive pigmentation is post-inflammatory pigmentation, where the skin darkens after an injury or inflammation has healed. Conditions like lichen planus, which causes purple, itchy bumps on the skin, can also lead to changes in skin color.[33] Essentially, reactive pigmented lesions are areas on the skin that have changed color due to some form of irritation or disease. These changes can affect anyone and may vary greatly in appearance, ranging from light brown to almost black, depending on the underlying cause and the individual's skin type.

Dermatofibroma

Dermatofibroma



Lichen aureus

Lichen planus

Our skin has a natural protector against the sun's harmful rays called eumelanin, a dark brown pigment that absorbs ultraviolet (UV) rays from the sun. This fascinating pigment plays a crucial role in determining the color of our skin. People with darker skin have more eumelanin, which gives their skin its rich hue, while people with lighter skin have less, leading to a paler complexion. Beyond just influencing skin color, eumelanin has significant health benefits.[34-38] It is particularly effective in reducing the risk of skin cancer. Research indicates that individuals with higher amounts of eumelanin in their skin are less likely to develop skin cancer compared to those with lower levels. This is because eumelanin can absorb UV radiation, which is a major cause of skin cancer. Moreover, eumelanin contributes to our body's ability to manage its temperature. By absorbing heat from the sun, it helps to keep our body cool, making it an essential component for regulating our overall body temperature.

Pheomelanin is a type of pigment that gives skin a lighter, yellowish-red color, which is different from the darker pigment known as eumelanin.[39] People who have more pheomelanin in their skin tend to have lighter skin shades and are more sensitive to the sun. This means they can get sunburned more easily because

pheomelanin isn't as good at blocking out harmful UV rays from the sun as eumelanin is. However, having pheomelanin also comes with its own advantages. For instance, it plays a role in regulating the body's temperature, especially in hot environments, by reflecting heat away from the skin.[40] Additionally, there's some evidence to suggest that pheomelanin might offer protection against certain kinds of skin cancer, including melanoma.

The production of melanin, which includes both eumelanin and pheomelanin, is influenced by a specific gene known as the MC1R gene. This gene is found on chromosome 16 at a location called q24.3.[41-45] It has a big impact on how our bodies react to sunlight, determining how easily we tan or get sunburned. It also affects the color of our skin and hair. Interestingly, the MC1R gene is also linked to an increased risk of developing melanoma, a type of skin cancer.

The amount of melanin that our bodies produce can vary quite a bit, even among members of the same family or people from different ethnic backgrounds. This variation is due to a number of factors, including genetics, how much sun exposure someone gets, and the influence of certain hormones that activate melanocytes, the cells responsible for making melanin.[46] These hormones include ACTH (adrenocorticotropic hormone), lipotropin, and melanocyte-stimulating hormone. People who produce more melanin tend to have a skin color that's more grayish-brown.

Causes:

Skin pigmentation, a frequent condition that affects many people, can result from several factors. Among the primary reasons for changes in skin color are genetic influences, the amount of sun the skin is exposed to, and the use of certain drugs.

our genetic makeup plays a significant role in determining our skin color and how susceptible we are to changes in pigmentation. Some people are naturally more prone to developing conditions like freckles, moles, or other changes in skin color

due to their genetic predisposition. Exposure to sunlight is a major factor that can affect our skin's pigmentation.[47] The sun's ultraviolet rays can trigger the production of melanin, the pigment that gives our skin its color. While a certain amount of sun exposure can lead to a healthy tan, too much sun can cause sunburn, dark spots, and other long-term changes in skin color.

certain medications can influence skin color by either increasing sensitivity to sunlight, which enhances melanin production, or by directly affecting the skin's pigmentation process. Some drugs known to cause changes in skin color include antibiotics, anti-inflammatory drugs, and medications for psychiatric conditions.

Research has discovered that a surprising number of genes, totaling 125, play a role in determining one's skin color.[48] The process through which our skin develops its color involves both genetics and hormonal influences, as outlined in scientific illustrations like Figure 1. This process is heavily influenced by the production of melanin, a key pigment in the skin. Individuals have the power to affect their skin's health and its pigmentation levels through choices like how much they expose themselves to sunlight or the use of various pharmaceuticals and cosmetic products. Over time, these decisions can lead to changes in skin color.

Genetics:

Genetics stand out as a primary factor influencing skin color, suggesting that it can determine the quantity of melanocytes, the cells responsible for melanin production, in each person. The role of melanocytes is crucial because they are the producers of melanin, which dictates the skin's color.[49-55] During certain conditions like hyperpigmentation or when the skin tans, the melanin-containing organelles, known as melanosomes, must be transported and increased in size. Conversely, in cases of hypopigmentation, these melanosomes diminish in size. Darker-skinned individuals naturally have more melanin compared to those with

lighter skin tones, which explains the variation in skin colors among different people.

sun exposure is another significant factor affecting skin pigmentation. When the skin is exposed to the sun, it produces more melanin as a form of protection against harmful ultraviolet (UV) rays.[56] This protective mechanism can result in a darker pigmentation to safeguard the skin from UV damage. The process through which sun exposure affects skin color involves several steps: UV radiation generates free radicals; these free radicals and UV light then stimulate certain biological agents that influence melanocytes, the pigment-producing cells. An enzyme called tyrosinase converts the amino acid tyrosine into melanin pigments, which are responsible for the skin's coloration. Various biological substances further enhance the activity of tyrosinase, leading to increased pigment production. As skin cells migrate to the surface and are naturally shed through exfoliation, melanin is lost from the skin. However, melanin granules from neighbouring keratinocytes (skin cells) are transferred to give the skin its distinctive color. This complex interplay between genetics, environmental factors, and individual choices underscores the dynamic nature of skin color determination.

Causes of hyperpigmentation:

Hyperpigmentation happens when the skin starts producing more melanin, which makes the skin darker in some areas. This can happen for many reasons, like being in the sun too much, having certain skin conditions, changes in hormones, getting older, genetic factors, skin damage, and even acne.[57] The sun is a big reason for this because it really increases melanin in the skin. Studies have shown that if you're in the sun a lot when you're young, it can make dark spots look worse, including spots from getting older or from inflammation. Hormonal changes, especially in women, can also cause hyperpigmentation, with conditions like chloasma and melasma. This is because the hormones oestrogen and progesterone

can make more melanin when the skin is exposed to the sun. Sometimes, treatments that change hormone levels can also make this condition worse.[58-60]

As people get older, the number of melanocytes (the cells that make melanin) goes down, but the ones that are left can get bigger and make more melanin, especially after you're over 40. Genetics also play a role in how much melanin your skin makes. After skin injuries or inflammation from things like chemical burns, wounds, or skin conditions like psoriasis, the skin can become darker in those areas, which is called post-inflammatory hyperpigmentation.[61] Even acne can lead to darker skin spots, especially if the acne is severe or if it's been picked at or popped. However, mild acne usually doesn't cause this problem. Certain things like pregnancy, age spots, scars from acne, and some medicines can also lead to hyperpigmentation. There's even a rare condition called Addison's disease that can cause dark patches on the skin because of issues with the adrenal glands. Sometimes, treatments using lasers or light can also cause the skin to become darker.[62-68]

Treatment:

Medications for treating skin pigmentation have gained popularity only in recent times, despite being known for many years. These treatments include both creams applied to the skin and pills taken orally, each with its own set of benefits and drawbacks. It's important to weigh these pros and cons to decide which treatment might be the best option.

Oral medications offer a promising alternative for addressing skin conditions and altering skin color. These pills are typically stronger than creams applied to the skin and require less frequent use. However, oral medications can be costly and might lead to more serious side effects compared to creams that you apply directly to the skin.[69]

Among the various treatments, tranexamic acid, a compound known for its ability to modify blood coagulation, stands out. Sold under the brand name Traxamac® 250 mg, tranexamic acid is utilized for a range of conditions including eczema, melasma, and more. It is recognized for its ability to alleviate symptoms like redness, itching, and swelling. The way tranexamic acid works is by blocking certain binding sites on molecules involved in blood clotting, which helps to prevent excessive bleeding. Recent research suggests that tranexamic acid can also interfere with the processes that lead to skin pigmentation by inhibiting an enzyme involved in melanin production. This makes it an effective option for preventing and treating hyperpigmentation, reducing the appearance of dark spots on the skin by decreasing the production of melanin.[70]

Creams applied directly to the skin are often used to address changes in skin color. They can either lighten or darken the skin, depending on the need, and are popular because they can be used comfortably at home. This convenience means no need to visit a doctor, and they usually cost less than pills taken by mouth. However, these creams have some downsides. Applying them can be messy and take a lot of time. Also, they might not work as well as the pills for some people.

Dermatologists frequently suggest using topical steroids for a range of skin issues, such as eczema, psoriasis, and other inflammatory conditions. These steroids work by reducing inflammation, slowing down cell growth, and suppressing the immune system to relieve symptoms. They are usually applied once to three times daily. Examples include betamethasone and clobetasol, which are known for their ability to inhibit certain body processes that lead to inflammation. They help in reducing the production of substances that can cause swelling and pain in the skin.[71] Despite their effectiveness, it's important to be cautious of both local and widespread side effects when using these steroids.

Natural Treatment:

Increasingly, people are turning towards plants and natural remedies as a preferred choice for treating various skin conditions, including hyperpigmentation. These traditional methods are becoming more popular for their perceived safety and effectiveness. Essential vitamins like A, B, C, and E play a crucial role in maintaining healthy skin and can be found in numerous foods and supplements.[72] Each of these vitamins provides unique benefits to the skin, helping to alleviate pigmentation issues.

Among the B vitamins, niacin (or niacinamide), pantothenic acid, and biotin stand out for their applications in skincare. Niacinamide, commonly included in facial creams, helps reduce the visibility of pores, fine lines, and dull skin. Pantothenic acid is beneficial for treating dry and flaky skin by acting as a moisturizer, while biotin is found in various products designed for healthier skin, nails, and hair. Vitamin C, a potent antioxidant, plays a significant role in preventing melanin synthesis by inhibiting certain processes that lead to hyperpigmentation. Research has demonstrated the effectiveness of vitamin C in reducing melanin production, thus aiding in skin lightening.[73]

Furthermore, extracts from plants such as Artocarpus lakoocha and Glycyrrhiza glabra have shown promise in reducing melanin pigment by targeting key factors involved in its production. Natural oils like rosehip, jojoba, and argan oil, known for their antioxidant and fatty acid content, can help reduce inflammation and brighten the skin. Aloe vera, renowned for its skin-lightening properties, serves as a non-toxic option for treating hyperpigmentation.

Jojoba oil is particularly valued for its moisturizing benefits without leaving a greasy residue, aiding in skin hydration and offering potential relief for conditions like dry skin and diaper rash. Argan oil has also been tested for its effects on skin pigmentation, with studies indicating its ability to reduce melanin content. Other natural ingredients such as licorice root extract, turmeric, and green tea extract have been acknowledged for their antioxidant, anti-inflammatory, and skin-

brightening properties.[74] These ingredients have been subject to various studies, showing their effectiveness in improving skin conditions and reducing hyperpigmentation.

Kojic acid is another compound that has gained attention for its ability to interfere with melanin production, offering a solution for dark spots and hyperpigmentation. It has been tested in clinical settings, demonstrating its potential in treating various skin conditions without causing adverse effects.

Moreover, marine-derived compounds and extracts from brown algae have been explored for their beneficial effects on skin health, including their role in reducing hyperpigmentation and preventing premature skin aging. These substances are noted for their antioxidant, anti-inflammatory, and skin-protective properties.

The growing interest in natural and plant-based treatments for skin hyperpigmentation reflects a broader trend towards safer and more holistic approaches to skincare.[75] Through ongoing research and clinical trials, the effectiveness of these natural remedies continues to be supported, offering promising alternatives for those seeking to improve their skin health naturally.

Modern Treatment:

Laser therapy has been a preferred choice for treating skin pigmentation issues for quite some time. This approach works by directing laser light at the skin to lighten and balance melanin, the pigment responsible for color in our skin. This process helps achieve a smoother and more uniform skin tone. Thanks to advancements in technology, lasers have become increasingly effective, capable of reaching and treating pigmentation beneath the skin's surface without harming the surrounding area. This means that issues like age spots and damage from the sun can be addressed more easily, without discomfort or adverse effects.[76] The latest laser technologies use a mix of light energy and intense pulsed light (IPL)

to tackle stubborn pigmentation, offering quicker and more efficient treatment options than ever before.

In addition to lasers, there are new treatments like topical creams and serums designed specifically to combat pigmentation. These products contain active ingredients such as niacinamide, kojic acid, licorice extract, and mulberry extract, which work together to fade dark spots, lighten the skin, and improve its texture and clarity. For example, kojic acid reduces the activity of tyrosinase, an enzyme involved in the production of melanin, thus preventing the formation of dark spots. Niacinamide helps in reducing skin discoloration by inhibiting melanin production.[77] It's important that these treatments are safe and do not contain harmful substances like parabens that could cause skin irritation.

Micro-needling is another innovative method that promotes the production of collagen and elastin, essential components for healthy skin. This procedure uses tiny needles to create microscopic channels in the skin, stimulating the body's healing process and leading to improved skin texture and a reduction in pigmentation. This method is gaining popularity due to its effectiveness and minimal downtime.

Chemical peels, which work by removing the outer layers of skin, also play a role in diminishing the appearance of dark spots. However, combining different treatments, such as various acids including glycolic and lactic acid, has been found to be more effective. These combination therapies offer benefits that surpass those of laser or light-based treatments alone, addressing facial discoloration and uneven skin tone in a single session.

Looking ahead, the prospects for treating skin pigmentation are bright, with new technologies offering more effective and less invasive options. Plasma pen therapy, for example, uses targeted plasma energy to treat freckles, age spots, and other forms of pigmentation with fewer side effects than traditional treatments.

Similarly, radiofrequency treatments, which use radio waves to break down melanin deposits, are becoming a popular option for improving the appearance of dark spots and enhancing the overall texture of the skin without requiring significant recovery time.[78,79] These advancements signal a future where treating skin pigmentation is more accessible, effective, and comfortable for patients.

Material & Methods

METHODOLOGY

- **Study Design**

The proposed research was designed as a hospital-based, cross-sectional observational study. This type of study allows for the collection and analysis of data at a specific point in time, providing a snapshot of the occurrence and characteristics of pigmented skin lesions within the study population. The observational nature of this study enabled researchers to observe the correlation between clinical and histopathological features of the lesions without any intervention or modification of the participants' conditions.

Place of Study

The study was conducted at the Pathology department of Santosh Medical College and Hospital, located in Ghaziabad.

Duration of Study

The study was conducted from August 2023 to December 2024.

Study Population

The study population consisted of both male and female individuals between the ages of 18 and 65 years. All the clinical details including age and sex, along with other relevant findings were taken from requisition forms received with skin biopsy samples.

Sample Size

The study involved a sample of 65 participants, each with one pigmented skin lesion. The sample size of 65 will ensure that the study has sufficient statistical power to detect meaningful correlations between the clinical features and histopathological findings of the lesions.

SUBJECT SELECTION

Inclusion Criteria

- Study will include patients who have pigmented skin lesions.
- The study will involve individuals from age groups 18-65 years, including both male and female population.

Exclusion Criteria

- Patients who have hypopigmented skin lesions.
- Any patients whose biopsy specimens are inadequate for analysis.
- Individuals who are not willing to provide a sample for the study.

SAMPLE SIZE n=65

Sample Size Determination

- The formula used for calculating the sample size is as follows:

$$n = \frac{Npq}{d^2/(1.96^2(N-1)) + pq}$$

-
- **n** represents the required sample size for the study.
- **N** is the total population size from which the sample will be drawn.

- **p** denotes the estimated proportion or prevalence of the characteristic being studied within the population.

q is the complement of p, calculated as $q=1-p$ $q = 1 - p$ $q=1−p$.

d is the desired level of absolute precision or margin of error, indicating the extent to which the sample estimate can differ from the true population parameter.

1.96 is a constant derived from the normal distribution, typically used for a 95% confidence interval, ensuring that the sample size calculation is appropriate for most studies.

- In reference to Schauffer RL, Mendenhall W, Ott L. Survey Sampling, Fourth edition. Duxberry press, Belmont California 1990.

Result

RESULT

Table 1: Distribution of study subjects according to age

Age	Frequency	% of Total
≤20	8	12.3 %
21-30	21	32.3 %
31-40	19	29.2 %
41-50	8	12.3 %
51-60	6	9.2 %
>60	3	4.6 %
Total	**65**	**100.0%**
Mean ± SD	33.5 ±12.4	

The above table represents the age distribution of the participants, with a total of 65 individuals. The highest frequency is observed in the 21-30 age group, comprising 32.3% of the total participants, followed by the 31-40 age group at 29.2%. The ≤20 and 41-50 age groups each account for 12.3%, while the 51-60 and >60 age groups represent 9.2% and 4.6%, respectively. The mean age of the participants is 33.5 years, with a standard deviation of 12.4 years, indicating a relatively wide age range within the study population.

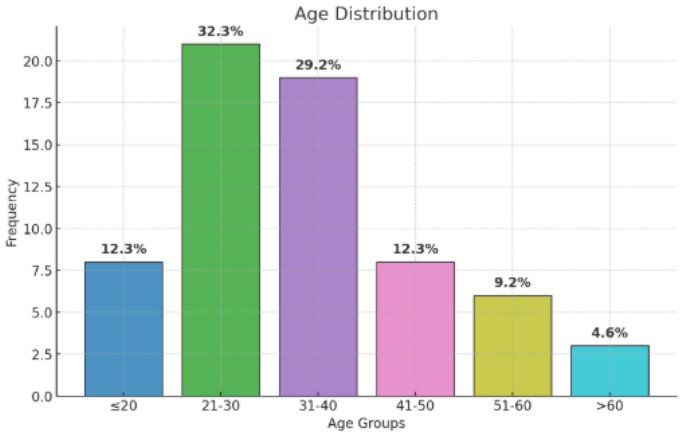


Table 2: Distribution of study subjects according to gender

GENDER	Frequency	% of Total
M	34	52.3 %
F	31	47.7 %
Total	**65**	**100.0%**

The above table represents the distribution of participants by gender in the study. Out of a total of 65 participants, 34 (52.3%) are male, and 31 (47.7%) are female, indicating a slightly higher proportion of male participants compared to females.

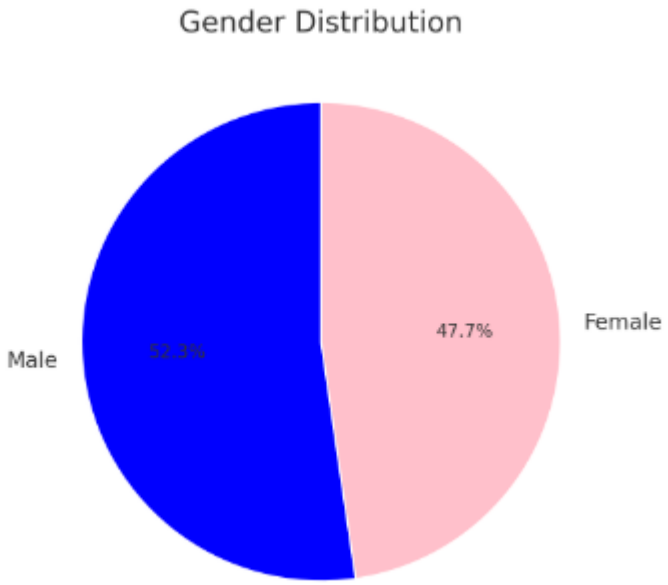


Table 3: Distribution of study subjects according to diagnosis

DIAGNOSIS	Frequency	% of Total
Lichen planus	33	50.7%
LP pilaris	1	1.5 %
LP simplex	1	1.5 %
LP chronicus	2	3.1 %
LP pigmentosus	9	13.8 %
Hypertrophic LP	5	7.6%
Nevus sebaceous	4	6.1 %
Basal cell carcinoma	1	1.5 %
Becker nevus	1	1.5 %
Prurigo Nodularis	3	4.6%
Prurigo Simplex	2	3.1%
Lichen Amylodosis	1	1.5%
Discoid lupus erythematous	1	1.5%
Seborrhic Keratosis	1	1.5%

The above table represents the distribution of various diagnoses in a sample group. Lichen planus (LP) was the most common diagnosis, accounting for 50.7% of the cases, followed by LP pigmentosus at 13.8%. Other forms of LP, such as LP chronicus and LP simplex, were reported at 3.1% and 1.5%, respectively. Hypertrophic LP was observed in 7.6% of the cases. Nevus sebaceous and Prurigo Nodularis were present in 7.7% and 4.6% of the patients, respectively. A number of rarer diagnoses, including Basal Cell Carcinoma,

Beckers Nevus, Prurigo Simplex, Lichen Amylodosis, Discoid Lupus Erythematous, and Seborrhic, were each noted in 1.5% of the cases.

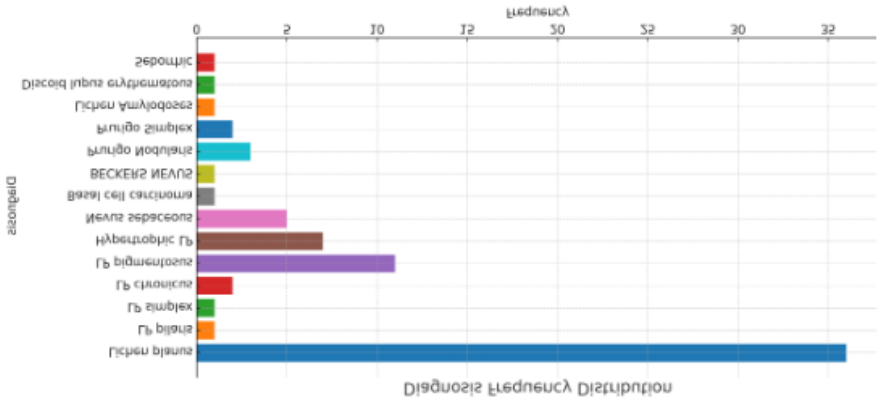


Table 4: Distribution of study subjects according to duration of lesion

Duration_of_Lesion	Frequency	% of Total
<6 months	20	30.8 %
1-2 years	24	36.9 %
Chronic	21	32.3 %
Total	**65**	**100.0%**

The above table represents the distribution of lesions based on their duration. Out of the total 65 cases, 20 (30.8%) had lesions lasting less than 6 months, 24 (36.9%) had lesions persisting for 1-2 year, and 21 (32.3%) had chronic lesions. This shows that the majority of lesions in the study had a duration of more than one year, followed by those with a lifelong chronic duration, and the least being those with a duration of less than 6 months.

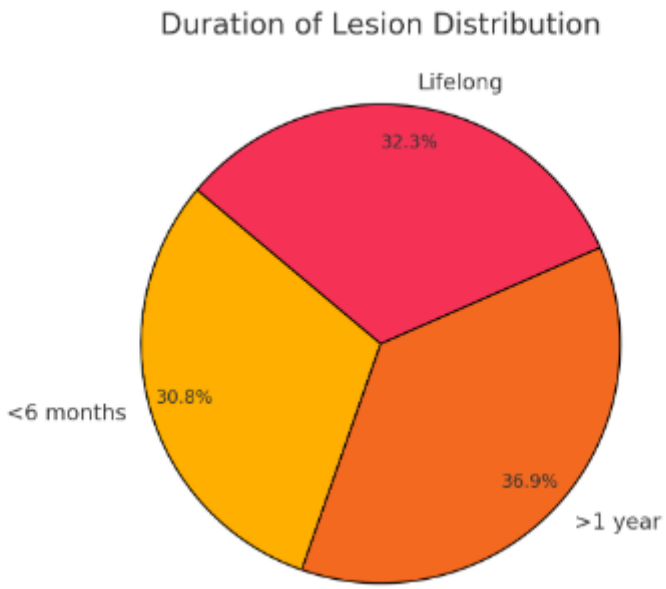


Table 5: Association Between Clinical Features and Their Presence/Absence in the Study Population

	Variables	Present	Absent	p value
Clinical features	**Itching**	64 (98.5%)	1 (1.5%)	<0.001
	Ulceration	20 (30.8%)	45 (69.2%)	0.002

The above table represents the distribution of clinical features in a study population, focusing on the presence of itching and ulceration. Itching was significantly prevalent, with 64 cases (98.5%) reported, and only 1 case (1.5%) absent, yielding a highly significant p-value of <0.001. On the other hand, ulceration was present in 20 cases (30.8%) and absent in 45 cases (69.2%), also showing statistical significance with a p-value of 0.002. These findings underscore the distinct clinical manifestations observed in the sample, highlighting the importance of these features in the study's context.

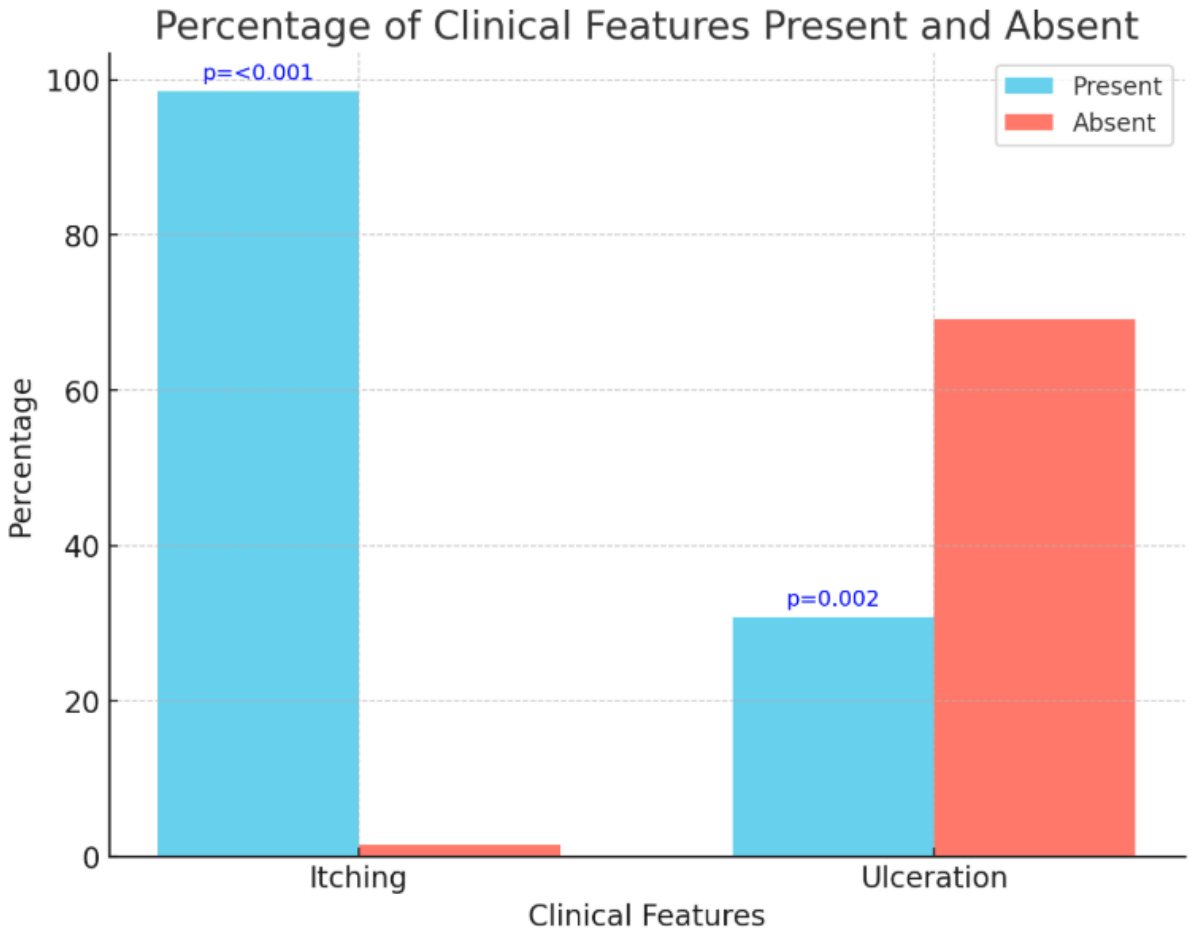


Table 6: Histopathological Features in Skin Lesions: Frequency of Presence and Absence with Associated p-Values

		Variables	Present	Absent	p value
Histopathological features	**Epidermis**	**Spongiosis**	18 (27.7%)	47 (72.3%)	<0.001
		Hyperkeratosis	44 (67.7%)	21 (32.3%)	0.004
		Parakeratosis	13 (20.0%)	52 (80.0%)	<0.001
		Acanthosis	45 (69.2%)	20 (30.8%)	0.002

		Max Joseph Space	42 (64.6%)	23 (35.4%)	0.018
		Saw-Toothed Rete Ridges	47 (72.3%)	18 (27.7%)	<0.001
		Hypergranulosis	47 (72.3%)	18 (27.7%)	<0.001
	Dermis	**Lymphocytic Infiltrate**	50 (76.9%)	15 (23.1%)	<0.001
		Perivascular Infiltration	17 (26.2%)	48 (73.8%)	<0.001
		Band Like Infiltrate	36 (55.4%)	29 (44.6%)	0.385
		Pigment Incontinence	50 (76.9%)	15 (23.1%)	<0.001

The above table represents the distribution and statistical significance of various histopathological features in the epidermis and dermis. In the epidermis, features such as spongiosis, hyperkeratosis, parakeratosis, acanthosis, Max Joseph Space, saw-toothed rete ridges, and hypergranulosis are evaluated, with the percentage

of presence and absence provided alongside their p-values. Notably, significant differences are observed in most features, with p-values less than 0.05, indicating statistical significance. For instance, hyperkeratosis is present in 67.7% of cases and absent in 32.3%, with a p-value of 0.004. Similarly, in the dermis, lymphocytic infiltrate and pigment incontinence are significantly present in 76.9% of cases, while perivascular infiltration shows a high percentage of absence. The band-like infiltrate did not show a significant difference, as indicated by a p-value of 0.385. This table succinctly outlines the prevalence and significance of each feature, highlighting the pathological characteristics observed in the study samples.

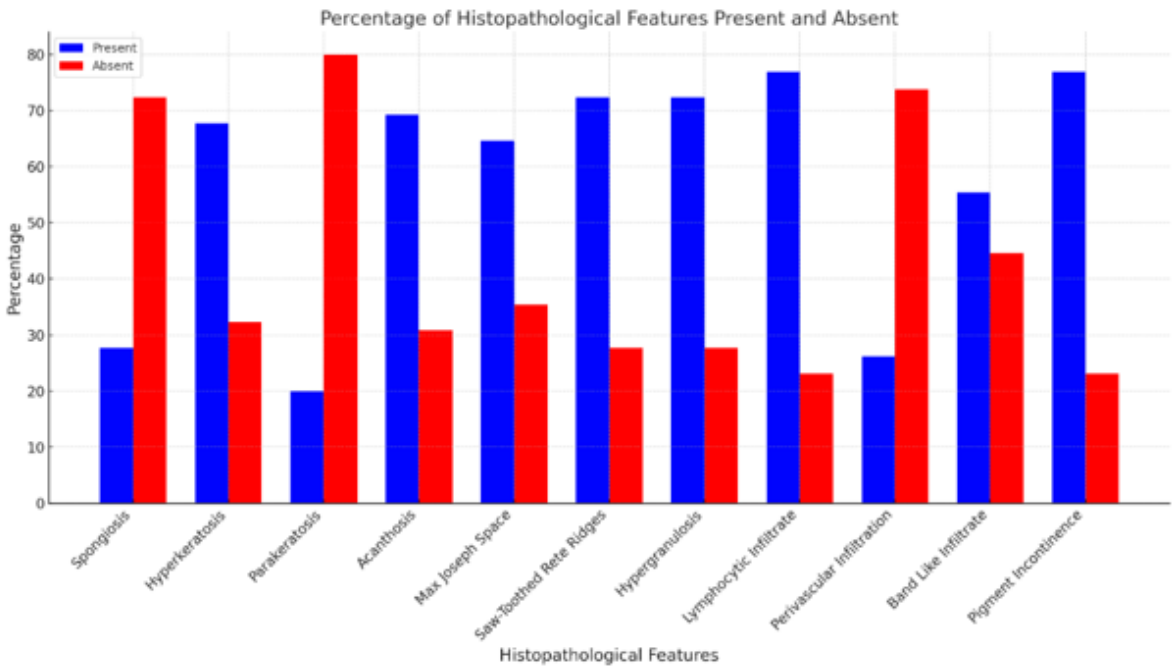
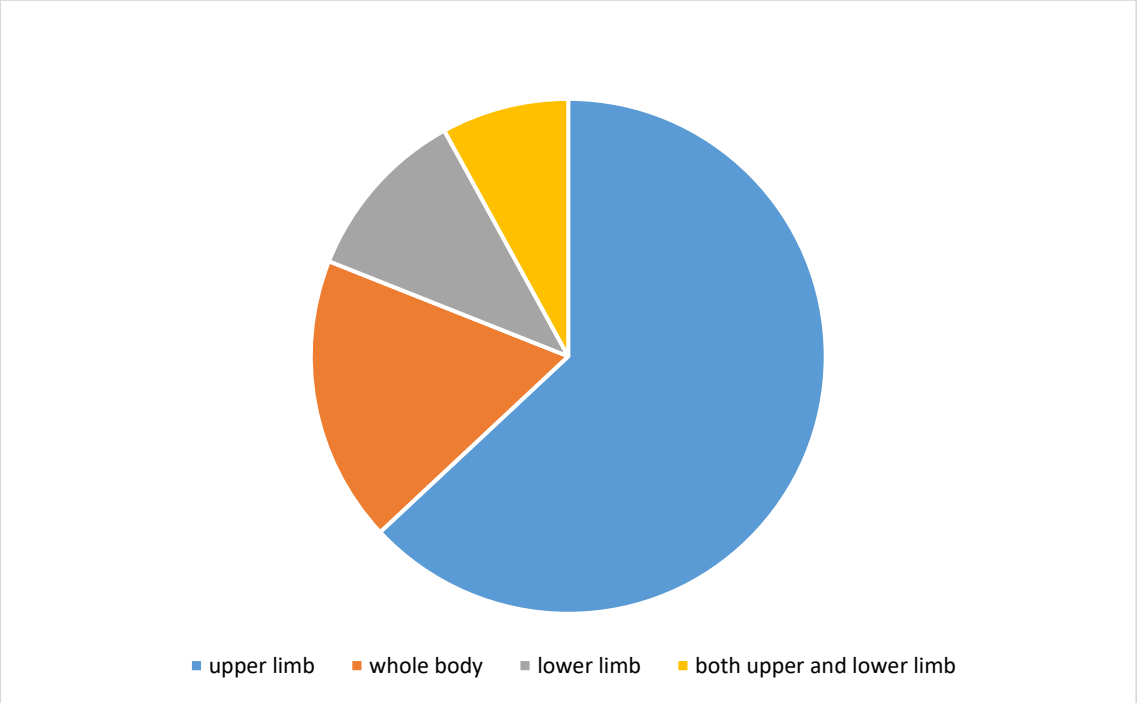


Table 7: Distribution of study subjects according to site distribution

Site Distribution	Frequency	% of Total
Upper limbs	41	63.1 %
Whole body	12	18.5 %
Lower limbs	7	10.8 %
Both upper and lower limbs	5	7.7 %
Total	**65**	**100.0%**



The above table represents the distribution of a certain condition based on different body sites. Among the total of 65 cases, the majority, 63.1%, are located in the upper limbs, followed by 18.5% of cases affecting the whole body. The lower limbs are involved in 10.8% of cases, while 7.7% of cases involve both the upper and lower limbs. This distribution highlights that the upper limbs are the most commonly affected site.

Table 8: Distribution of study subjects according to presentation type

Presentation Type	Frequency	% of Total
Plaques	23	35.4 %
Patches	2	3.1 %
Papules	36	55.4 %
Macules	4	6.2 %
Total	**65**	**100.0%**

The above table represents the distribution of different presentation types among the 65 total cases. The majority of the cases were characterized by papules, accounting for 55.4% (36 cases). Plaques were observed in 35.4% (23 cases), followed by macules, which made up 6.2% (4 cases). The least frequent presentation type was patches, representing 3.1% (2 cases). Overall, the total number of cases across all categories sums to 100%.

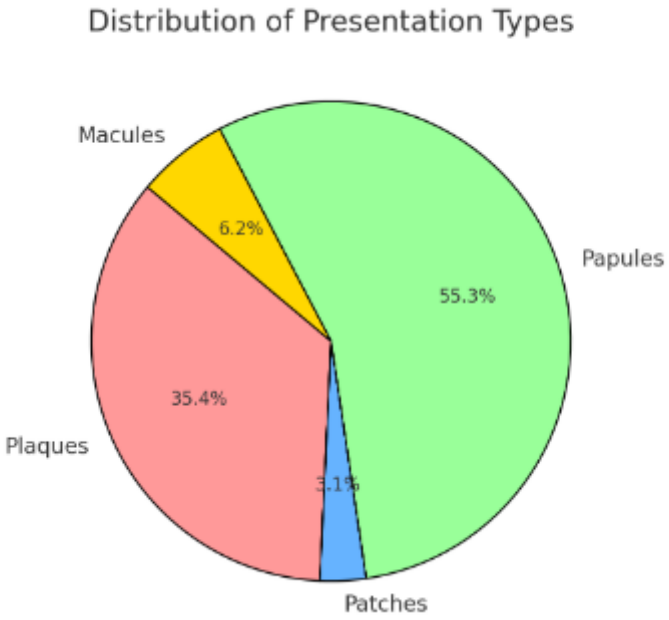


Table 9: Distribution of study subjects according to clinical findings

Clinical_Findings	Frequency	% of Total
Violaceous papules and plaques, Itching	36	55.4 %
Thickened plaques with chronic itching	2	3.1 %
Hyperpigmented patch, sometimes with hair growth	1	1.5 %
Localized pruritic plaques, Chronic itching	1	1.5 %
Hyperpigmented macules/patches, mild itching	11	16.9 %
Thickened hyperkeratotic plaques, chronic itching	7	10.8 %
Patch or Hairless area	5	7.7 %
Follicular papules, itching	1	1.5 %
Other clinical findings	1	1.5 %
Total	**65**	**100.0%**

The above table represents the distribution of various clinical findings observed in the study. The most common clinical finding is violaceous papules and plaques associated with itching, accounting for 55.4% of the total cases. Other significant findings include thickened plaques with chronic itching (3.1%), hyperpigmented patches with occasional hair growth (1.5%), localized pruritic plaques with chronic itching (1.5%), and hyperpigmented macules or patches with mild itching (16.9%). Additionally, thickened hyperkeratotic plaques with chronic itching were noted in 10.8% of cases, while patch or hairless areas and follicular papules

with itching each contributed to 1.5%. Lastly, there were other clinical findings in 1.5% of the cases, making up the remainder of the observations.

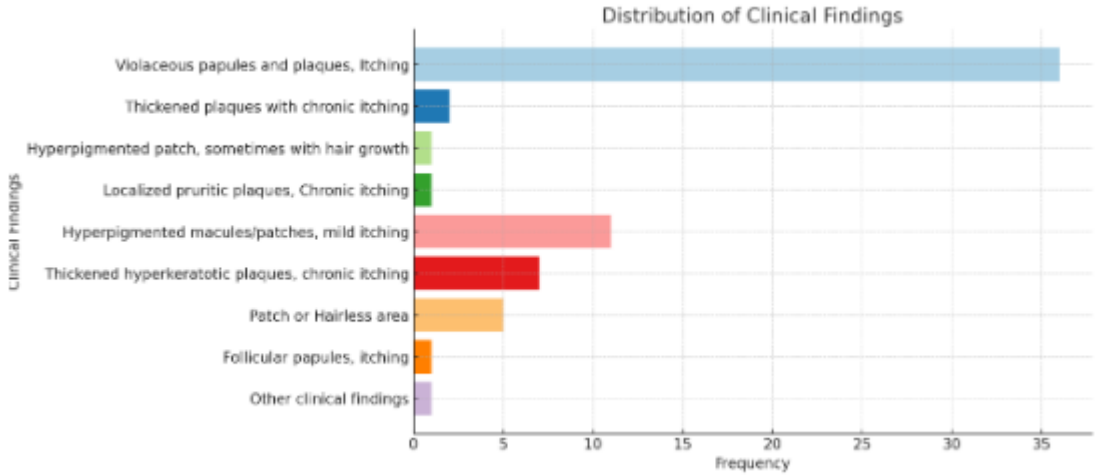


Table 10: Distribution of study subjects according to histopathological findings

Histopathological_Findings	Frequency	% of Total
Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space	36	55.4 %
Epidermal hyperplasia, Hypergranulosis, Chronic inflammation	2	3.1 %
Hyperpigmentation, Occasional smooth muscle proliferation, Acanthosis	1	1.5 %
Thickened epidermis, Minimal inflammation	1	1.5 %
Basal layer hyperpigmentation, sparse lymphocytic infiltrate	11	16.9 %

Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate	7	10.8 %
Hyperkeratosis, sebaceous gland hyperplasia	5	7.7 %
Follicular plugging, hyperkeratosis, lymphocytic infiltrate	1	1.5 %
Other histopathological findings	1	1.5 %
Total	**65**	**100.0%**

The above table represents the distribution of various histopathological findings in the studied samples. The most common finding was hyperkeratosis, acanthosis, band-like lymphocytic infiltration, and Max Joseph space, observed in 36 cases (55.4%). Other notable findings included basal layer hyperpigmentation with sparse lymphocytic infiltrate (16.9%) and hyperkeratosis with epidermal hyperplasia and chronic inflammatory infiltrate (10.8%). Additionally, epidermal hyperplasia, hypergranulosis, and chronic inflammation were seen in 3.1% of cases, while less frequent findings such as hyperpigmentation, smooth muscle proliferation, and follicular plugging were also observed. The table highlights the diverse range of histopathological patterns, with 65 cases in total, making up 100% of the sample.

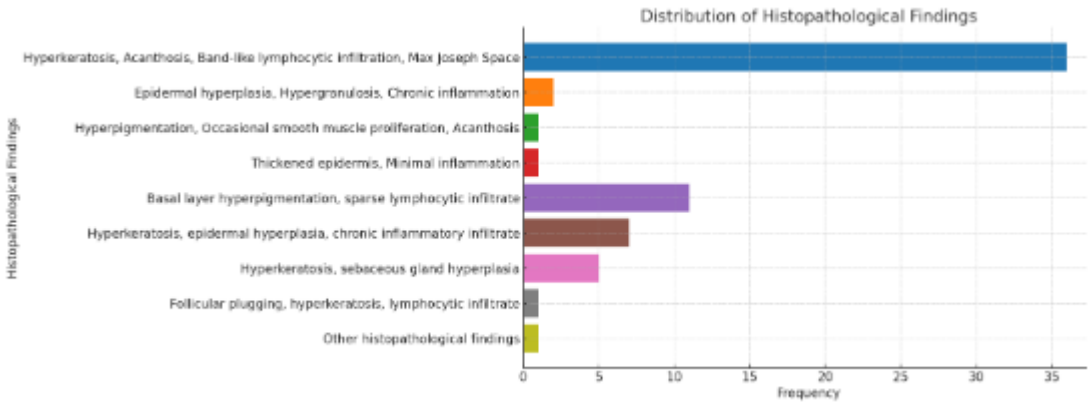


Table 11: Association of presentation type & site distribution

Presentation Type	Upper limbs	Whole body	Lower limbs	Both upper and lower limbs	Total
Plaques	13 (56.5%)	10 (43.5%)	0 (0.0%)	0 (0.0%)	23 (35.4%)
Patches	2 (100.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (3.1%)
Papules	24 (66.7%)	0 (0.0%)	7 (19.4%)	5 (13.9%)	36 (55.4%)
Macules	2 (50.0%)	2 (50.0%)	0 (0.0%)	0 (0.0%)	4 (6.2%)
Total	41 (100.0%)	12 (100.0%)	7 (100.0%)	5 (100.0%)	65 (100.0%)

The above table represents the distribution of various skin lesion types across different body regions. Plaques were most commonly observed on the upper limbs (56.5%) and whole body (43.5%), accounting for 35.4% of all cases. Papules were the most frequent overall, with 66.7% occurring on the upper limbs, 19.4% on the lower limbs, and 13.9% on both upper and lower limbs, making up 55.4% of the total cases. Patches were exclusively found on the upper limbs (100%), contributing to 3.1% of the total cases. Macules were equally distributed between the upper limbs and the whole body (50% each), representing 6.2% of all cases. In total, the study included 65 cacases, with the majority presenting on the upper limbs.

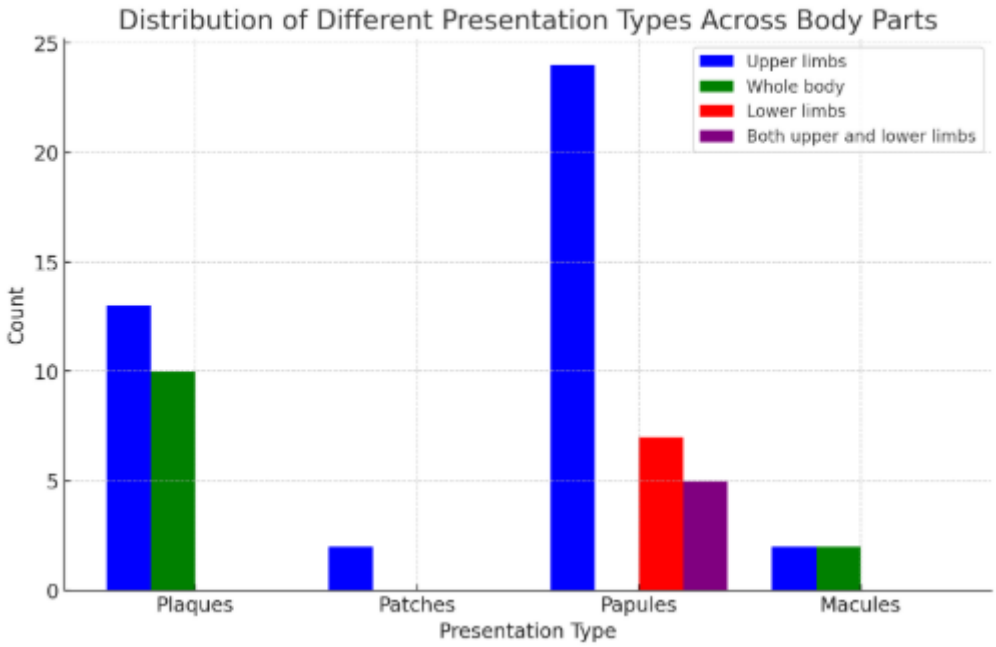
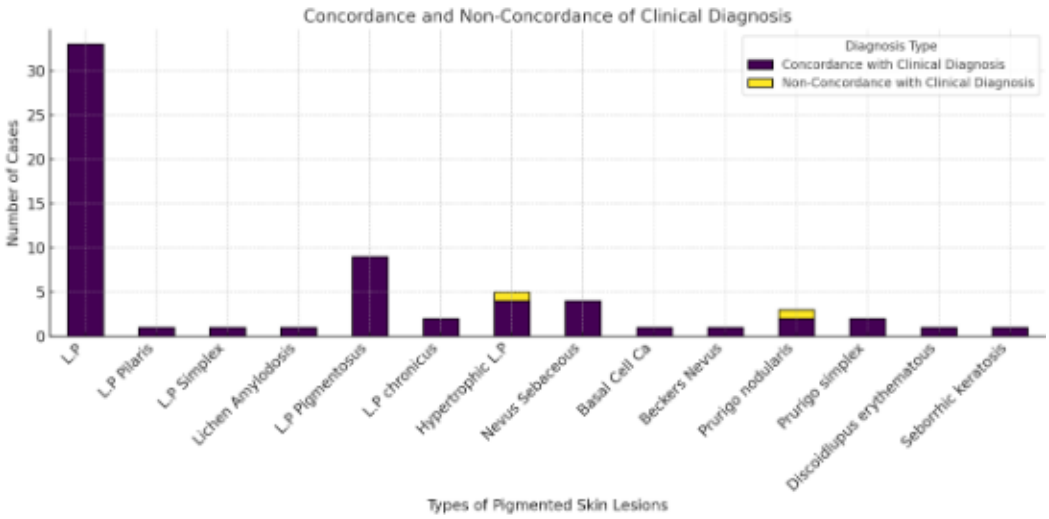


Table 12: Histopathological Diagnosis

Types of pigmented skin lesions	**No. of cases**	**Concordence with clinical Diagnosis**	**Non-Concordence with clinical Diagnosis**
L.P	33	33	Nil
L.P Pilaris	1	1	Nil
L.P Simplex	1	1	Nil
Lichen Amylodosis	1	1	Nil
L.P Pigmentosus	9	9	Nil
L.P chronicus	2	2	Nil
Hypertrophic L.P	5	4	1
Nevus Sebaceous	4	4	Nil
Basal Cell Ca	1	1	Nil
Beckers Nevus	1	1	Nil
Prurigo nodularis	3	2	1
Prurigo simplex	2	2	Nil

Discoid lupus erythematous	1	1	Nil
Seborrhic keratosis	1	1	Nil

The above table represents various types of pigmented skin lesions and their concordance with clinical diagnosis. Out of 33 cases of Lichen Planus (L.P.), all were in concordance with the clinical diagnosis. Similarly, other types like L.P. Pilaris, L.P. Simplex, Lichen Amylodosis, L.P. Pigmentosus, L.P. Chronicus, Nevus Sebaceous, Basal Cell Carcinoma, Becker's Nevus, and Discoid Lupus Erythematous showed complete concordance. However, some lesions such as Hypertrophic L.P. (with 1 case showing non-concordance) and Prurigo Nodularis (with 1 case of non-concordance) had minor discrepancies. Prurigo Simplex showed full concordance. Seborrheic Keratosis, like most other lesions, showed perfect concordance as well.



Discussion

DISCUSSION

The present study was conducted as a hospital-based cross-sectional observational study at the Pathology departments of Santosh Medical College and Hospital, Ghaziabad. The study aimed to establish a correlation between the histopathological and clinical features of pigmented skin lesions to enhance diagnostic accuracy. The study included both male and female participants aged 18 to 65 years who attended the Dermatology Department with clinically diagnosed pigmented skin lesions. Histopathological evaluation plays a crucial role in differentiating benign from malignant pigmented lesions and provides definitive confirmation of clinical diagnoses. The study sought to assess the histopathological spectrum of pigmented lesions, ranging from benign melanocytic nevi and lentigines to malignant melanoma and other pigmented skin disorders. By analyzing tissue biopsies and correlating them with clinical presentations, the study aimed to highlight the significance of histopathological examination as a gold standard for diagnosing pigmented skin lesions. Furthermore, the study emphasized the importance of early and accurate diagnosis, which is critical for effective patient management and treatment planning. The findings were expected to provide insights into the prevalence and histopathological variations of pigmented skin lesions in the studied population. Additionally, the study aimed to contribute to the existing knowledge regarding the clinical and pathological characteristics of these lesions, thereby assisting dermatologists and pathologists in improving diagnostic precision.

The present study aimed to establish a histopathological and clinical correlation of pigmented skin lesions in patients attending the Dermatology Department of Santosh Medical College and Hospital, Ghaziabad. A total of 65 cases were analyzed, covering a wide age range from 18 to 65 years. The mean age of the participants was 33.5 ± 12.4 years, indicating that pigmented skin lesions were more commonly observed in younger adults. The highest proportion of cases was

in the 21-30 years age group (32.3%), followed by the 31-40 years group (29.2%), suggesting that these lesions are more prevalent in early to mid-adulthood. In contrast, older age groups, particularly those above 60 years, had a lower incidence (4.6%). In terms of gender distribution, males (52.3%) slightly outnumbered females (47.7%), indicating a nearly equal prevalence of pigmented skin lesions across genders. These findings align with previous studies where no significant gender predilection was observed, although some literature suggests a slightly higher frequency of pigmented skin conditions in females due to hormonal influences and cosmetic concerns leading to higher healthcare-seeking behavior. The most common histopathological diagnosis in our study was lichen planus (LP)**,** which accounted for 50.7% of cases. Among the different variants of lichen planus, LP pigmentosus (13.8%) and hypertrophic LP (7.6%) were the most frequently observed subtypes. The predominance of LP in our study aligns with existing dermatopathology research, where LP and its variants are commonly associated with hyperpigmentation and chronic inflammatory changes. The presence **of** LP pilaris (1.5%), LP simplex (1.5%), and LP chronicus (3.1%) further highlights the clinical diversity of this condition. Apart from LP, nevus sebaceous (6.1%) was another frequently observed condition. This congenital hamartomatous lesion is often benign but may have potential for secondary neoplastic changes, emphasizing the importance of histopathological evaluation. Basal cell carcinoma (1.5%)**,** though rare in our study, underscores the necessity of biopsy for suspected malignant pigmented lesions, as early detection significantly impacts prognosis. Other conditions such as Becker's nevus (1.5%), prurigo nodularis (4.6%), prurigo simplex (3.1%), lichen amyloidosis (1.5%), discoid lupus erythematosus (1.5%), and seborrheic keratosis (1.5%) were observed in smaller proportions. Ravindran S et al.[80] analysed the distribution of hyperpigmented skin lesions across different age groups and sexes. The findings revealed that hyperpigmentation was more prevalent among females (53%) compared to males (47%). The most affected age

group was 21–30 years, accounting for 43% of the cases, followed by the 31–40 and >50 age groups, each contributing 15% to the total cases. The lowest prevalence was observed in the birth–10 years category, representing only 3% of the cases. These results suggest that hyperpigmentation is more commonly seen in young adults, possibly due to increased exposure to environmental factors, hormonal influences, and occupational exposures. When categorizing the different hyperpigmented skin lesions, classical lichen planus emerged as the most common condition, affecting 24% of the study population. Eczematous dermatitis was the second most frequent lesion, accounting for 12% of cases, followed by polymorphous light eruption (8%) and discoid lupus erythematosus (7%). Other conditions such as lichenoid-like keratosis and reaction (7%)**,** post-inflammatory hyperpigmentation (7%)**,** and lichen planus pigmentosus (6%) were also observed in notable proportions. Less frequent lesions included lichen amyloidosis (2%)**,** erythema dyschromium perstans (1%)**,** and prurigo nodularis (3%)**,** indicating that these conditions are relatively rare. The predominance of classical lichen planus aligns with previously reported studies highlighting its higher occurrence among individuals with chronic inflammatory dermatoses. The presence of post-inflammatory hyperpigmentation and lichenoid-like keratosis suggests that many cases of hyperpigmentation might be secondary to inflammatory dermatoses rather than primary pigmentary disorders. Furthermore, the occurrence of discoid lupus erythematosus (7%) and morphea (4%) highlights the relevance of autoimmune processes in hyperpigmentation pathogenesis. The study emphasizes the need for early diagnosis and intervention, especially in younger individuals who are more commonly affected. Preventive measures such as sun protection, early treatment of inflammatory skin conditions, and lifestyle modifications could play a crucial role in reducing the burden of hyperpigmented lesions. Further studies with larger sample sizes and histopathological correlations would help in better understanding the underlying mechanisms contributing to hyperpigmentation disorders.

PREVALENCE OF CASES AMONG VARIOUS STUDIES

Present study	Ravindran S et al [80]	Mruthyunjayappa S et al [81]	Jaiswal A et al [82]	Jagannadhan R et al [83]
• Lichen planus and its variants • Nevus • Sebaceous • Basal Cell Carcinoma • Becker's Nevus • Prurigo Nodularis • Prurigo Simplex • Lichen Amyloidosis • Discoid Lupus Erythematosus • Seborrheic Keratosis	• Classical Lichen Planus • Polymorphous Light Eruption • Discoid Lupus erythematous • Post-inflammatory Hyperpigmentation • Lichen Planus Pigmentosus • Lichen Amyloidosis • Prurigo Nodularis	• Classical lichen planus • Hypertrophic lichen planus • Lichenoid drug eruption • Lichen planus pigmentosus • Lichen simplex chronicus • Post- inflammatory hyperpigmentation • Prurigo nodularis	• Classical Lichen Planus • Polymorphous Light Eruption • Discoid Lupus Erythematosus • LichenPlanus Hypertrophicus • Lichenoid like keratosis and reaction • Lichen Planus Pigmentosus • Lichen Simplex Chronicus • Prurigo Nodularis • Lichen Amyloidosis	• Lichen planus and its variants including lichenoid keratosis • Benign melanocytic nevi • Seborrheic keratosis • Basal cell carcinoma • Malignant melanoma

Present study showed Lichen Planus and its variants as the most common hyperpigmented skin lesions diagnosed. This diagnosis was in concordance with various studies which showed Lichen Planus as the most common diagnosis.

In our study, nearly all subjects (98.5%) reported itching, making it the most common clinical feature observed. This high prevalence (64 out of 65 cases) was statistically significant ($p < 0.001$) and suggests that pruritus is an important indicator of pigmented skin lesions. In contrast, ulceration was present in about 31% of cases (20 cases), with a p-value of 0.002, highlighting that while less frequent than itching, ulceration still plays a notable role in the clinical presentation of these lesions.

Histopathologically, several key epidermal changes were identified. Spongiosis was observed in 27.7% of cases (18 cases), an indication of intercellular edema likely due to inflammatory processes, with a highly significant association ($p < 0.001$). Hyperkeratosis and acanthosis were even more common, seen in 67.7% (44 cases) and 69.2% (45 cases) of lesions respectively, with both findings reaching statistical significance ($p = 0.004$ for hyperkeratosis and $p = 0.002$ for acanthosis). Parakeratosis, noted in 20% of cases (13 cases), also demonstrated a significant relationship ($p < 0.001$). Additionally, Max Joseph space was present in 64.6% of cases (42 cases) with a p-value of 0.018, while both saw-toothed rete ridges and hypergranulosis were identified in 72.3% of cases (47 cases each), each with p-values below 0.001. These features together underscore the typical epidermal alterations seen in pigmented lesions. Turning to the dermis, lymphocytic infiltrate and pigment incontinence were among the most frequently observed features, each present in 76.9% of cases (50 cases) with p-values less than 0.001, emphasizing the role of immune response and melanin migration in these lesions. Perivascular infiltration was found in 26.2% of cases (17 cases) and was statistically significant ($p < 0.001$), whereas a band-like infiltrate was noted in 55.4% of cases (36 cases) but did not show a significant association ($p = 0.385$).

Mruthyunjayappa S et al.[81] evaluated five conditions: lichen planus, hypertrophic lichen planus, lichenoid drug eruptions, lichen planus pigmentosus, and discoid lupus erythematosus.

The histopathological examination revealed both overlapping and distinct features across these disorders. For instance, hyperkeratosis—an abnormal thickening of the outermost skin layer—was noted in about 71% of lichen planus cases, reached 100% in both hypertrophic lichen planus and discoid lupus erythematosus, yet was present in only 18% of lichen planus pigmentosus lesions. This suggests that a robust keratin response is more typical in conditions with a more prolonged inflammatory stimulus. When looking at the epidermis further, parakeratosis, which indicates retained nuclei in the stratum corneum, was infrequent in lichen planus (6%) but much more common in hypertrophic lichen planus (42%), possibly reflecting a higher rate of epidermal turnover in the latter. Similarly, acanthosis—representing a thickened epidermis—was found in 26% of lichen planus cases and in half of both hypertrophic lichen planus and lichenoid drug eruptions, while only 20% of lichen planus pigmentosus lesions showed this change. Hypergranulosis, the thickening of the granular cell layer, appeared in 30% of lichen planus and 50% of lichenoid drug eruptions, reinforcing its role as a classical finding in these diseases. A consistently striking feature across nearly all conditions was basal cell degeneration. This change, which disrupts the normal architecture of the skin's basal layer, was present in 100% of lichen planus, hypertrophic lichen planus, lichenoid drug eruptions, and discoid lupus cases, and in 87% of lichen planus pigmentosus lesions. Such universal involvement underscores the central role of interface dermatitis in these disorders. In addition, epidermal thinning was noted in 17% of lichen planus cases, in nearly half (47%) of lichen planus pigmentosus, and in all discoid lupus cases, indicating that prolonged damage may lead to an atrophic epidermis. Other features such as Civatte bodies (apoptotic keratinocytes) were identified in roughly a quarter of

lichen planus and hypertrophic lichen planus cases, while Max Joseph's space and saw-toothed rete ridges were less frequent findings. Notably, follicular plugging was a feature unique to discoid lupus, seen in every case examined, and correlates with its clinical presentation involving scarring and hair loss. The dermal examination provided further insight into the inflammatory processes at work. A band-like lymphocytic infiltrate was a hallmark of both lichen planus and hypertrophic lichen planus, present in all cases studied, which is typical for an interface dermatitis reaction. A dense lymphocytic infiltrate was observed in 90% of lichen planus cases and in every discoid lupus case, emphasizing the immune-mediated nature of these lesions. Perivascular infiltrates were common to all conditions—with 80% of lichen planus, 83% of hypertrophic lichen planus, and 100% of lichenoid drug eruptions, lichen planus pigmentosus, and discoid lupus cases showing this pattern—highlighting the role of blood vessels in the inflammatory cascade. In discoid lupus, the presence of perifollicular lymphocytes in all cases further points to the involvement of hair follicles, a feature that can explain the scarring seen clinically. Pigment incontinence, where melanin is lost from damaged basal cells into the dermis, was noted in 80% of lichen planus cases and was universally present in lichen planus pigmentosus, while being less prominent in hypertrophic lichen planus (25%) and moderate in lichenoid drug eruptions (50%). Finally, the exclusive finding of eosinophils and neutrophils in all cases of lichenoid drug eruptions suggests a distinctive allergic or toxic reaction underlying these lesions.

Feature	Cases (n)	Percentage
Clinical Features		
Itching	64	98.5%
Ulceration	20	31.0%

Histopathological Features (Epidermis)	Present study	Shushan Shweta Jayker et al[84]
Hyperkeratosis	67.7%	63.6%
Spongiosis	27.7%	3.6%
Parakeratosis	20%	23.6%%
Acanthosis	69.2%	49.5%
Max Joseph Space	64.6%	-
Saw-Toothed Rete Ridges	72.3%	-
Hypergranulosis	72.3%	15.3%
Histopathological Features (Dermis)		
Lymphocytic Infiltrate	76.9%	10.6%
Perivascular Infiltration	26.2%	40%
Band Like Infiltrate	55.4%	30.6%
Pigment Incontinence	76.9%	76.9%

In our study we observed that most common epidermal changes were hyperkeratosis, acanthosis, max-joseph space, saw tooth rete ridges, Hypergranulosis and dermal changes were lymphocytic infiltration, pigment incontinence.

Sushan Shweta Jayker et al[84], observed epidermal changes which were hyperkeratosis(63.6%) and acanthosis(49.2%). Dermal changes were pigment incontinence(76.9%) and perivascular infiltration(40%). These were the most common microscopic findings of the study. This study shows much similarity with our study as we found epidermal changes like hyperkeratosis(67.7%) and

acanthosis(69.2%) and dermal changes like pigment incontinence(76.9%) in common.

In our study, the duration of pigmented skin lesions among 65 patients varied considerably. Specifically, 20 patients (30.8%) had lesions that appeared within the past six months, indicating a group with relatively recent changes. In contrast, 24 patients (36.9%) reported that their lesions had persisted for 1-2 years, suggesting a slightly higher prevalence of long-standing lesions. Additionally, 21 patients (32.3%) indicated that the lesions had been present for as long as they could remember, reflecting a chronic occurrence. This nearly even distribution across the three categories underscores the diverse clinical presentation of pigmented skin lesions in our cohort. The variation in lesion duration is particularly important as it can influence diagnostic considerations; newer lesions may warrant prompt investigation due to the potential for rapid change, while chronic or lifelong lesions might be monitored differently based on their stability and history. Overall, these findings emphasize the need for a thorough clinical evaluation and histopathological correlation to tailor management strategies effectively for different lesion durations. Any relevant study which relates to Distribution of study subjects according to duration of lesion.

In our study, a total of 65 subjects were examined for the distribution of pigmented skin lesions across various body sites. Notably, the upper limbs were the most commonly affected area, with 41 individuals (63.1%) showing lesions in this region. This high prevalence might be related to greater exposure to environmental factors such as sunlight, which is known to influence pigmentation. Lesions involving the entire body were observed in 12 cases (18.5%), suggesting a more generalized pattern in a subset of patients. In contrast, the lower limbs were affected in 7 subjects (10.8%), while a smaller group of 5 individuals (7.7%) had lesions present on both the upper and lower limbs. These findings underscore the importance of focusing preventive and diagnostic efforts

on areas most susceptible to pigmentation changes, particularly the upper limbs, and prompt further investigation into the factors that contribute to the varied distribution of these lesions. Jaiswal A et al.[82] observed that 25% of cases involved only the upper limbs, while 9% were confined to the lower limbs. Interestingly, nearly half of the cases (44%) affected both the upper and lower limbs simultaneously, indicating that injuries were often not isolated to a single region. Additionally, 20% of cases were spread over the whole body, encompassing areas such as the chest, abdomen, back, and limbs, and a smaller fraction, 2%, involved the head, face, and neck. These findings suggest that the mechanism of injury or exposure likely resulted in more widespread trauma rather than isolated injuries. The predominance of cases affecting multiple limb regions could imply that the forces involved were distributed over a larger area, necessitating comprehensive evaluation and management. This pattern emphasizes the need for treatment approaches that are adaptable to both localized and more diffuse injuries, aligning with current evidence that multi-regional involvement often demands more integrated care strategies

Body Site	**Study Subjects**	**Study Percentage**	**Jaiswal A et al[82] Percentage**
Upper Limbs	41	63.1%	25%
Entire Body	12	18.5%	20%
Lower Limbs	7	10.8%	9%
Both Upper and Lower Limbs	5	7.7%	44%
Head, Face, and Neck	Not reported	Not reported	2%

In our study of 65 patients with pigmented skin lesions, the clinical evaluation revealed that papules were the most common presentation, observed in 55.4% of the cases (36 patients). Plaques followed, seen in 35.4% (23 patients), while

macules and patches were much less frequent, noted in 6.2% (4 patients) and 3.1% (2 patients), respectively. This distribution suggests that most pigmented lesions tend to present as raised or slightly elevated forms rather than flat discolorations, which could influence the approach to clinical diagnosis. Alongside the clinical findings, we also assessed the application of special stains during histopathological examination. Most cases (72.3%) did not require additional staining beyond routine methods. However, in cases where the routine staining did not provide clear diagnostic details, special stains were applied selectively: the PAS stain was used in 7.7% of cases, Crystal Violet in 10.8%, and Fontana Masson in 9.2% of the subjects. This targeted use of special stains underscores their importance in clarifying histopathological features when necessary, ultimately supporting a more precise diagnosis and better patient management. We did not find any relevant studies which relates to Distribution of study subjects according to presentation type and Distribution of study subjects according to special stains.

In our study of 65 patients with pigmented skin lesions, we observed a strong correlation between clinical presentations and histopathological findings. More than half of the subjects (55.4%) presented with violaceous papules and plaques associated with itching, and their biopsies consistently revealed features such as hyperkeratosis, acanthosis, a band-like lymphocytic infiltration, and the presence of Max Joseph spaces. Similarly, patients who exhibited hyperpigmented macules or patches with mild itching (16.9%) showed a corresponding pattern of basal layer hyperpigmentation along with a sparse lymphocytic infiltrate. Less common clinical features, such as thickened plaques with chronic itching (3.1%) and thickened hyperkeratotic plaques (10.8%), were matched by histological findings including epidermal hyperplasia with hypergranulosis or a chronic inflammatory infiltrate. Additionally, unique clinical presentations like localized pruritic plaques, hyperpigmented patches sometimes associated with hair growth,

patch or hairless areas, and follicular papules (each accounting for 1.5–7.7% of cases) were reflected in the histopathology by minimal inflammation, occasional smooth muscle proliferation, sebaceous gland hyperplasia, and follicular plugging, respectively. This consistent clinico-pathological correlation highlights the importance of histopathological examination in confirming clinical diagnoses and guiding appropriate management for pigmented skin lesions. We did not find any relevant studies which relates to Distribution of study subjects according to clinical findings and Distribution of study subjects according to histopathological findings.

In our study, the histopathological examination of pigmented skin lesions revealed a variety of conditions, with a majority of cases showing a high concordance with the clinical diagnoses. The types of pigmented lesions included lichen planus (LP) in various forms, sebaceous nevi, basal cell carcinoma, and others. Among the 65 cases studied, lichen planus (LP) and its subtypes were the most prevalent, with 33 cases of LP showing full concordance with clinical diagnosis. This suggests that LP, a common inflammatory disorder, is often easily recognizable based on both clinical and histopathological features. Other lesions, such as LP pigmentosus, also showed full concordance with clinical diagnoses, with nine cases being correctly identified. In contrast, hypertrophic lichen planus showed one case of non-concordance, where histopathology did not align with clinical findings, indicating a potential area for further investigation in differentiating this condition from others based solely on clinical presentation. Similarly, prurigo nodularis exhibited two cases of non-concordance, highlighting the challenges in distinguishing this lesion from other pigmented skin disorders clinically. Seborrheic keratosis, basal cell carcinoma, and Becker's nevus all showed complete concordance with clinical diagnoses. This finding reinforces the reliability of clinical examination for these specific conditions. Additionally, conditions like prurigo simplex and discoid lupus erythematosus

also demonstrated full concordance, reinforcing the role of histopathology as an essential diagnostic tool in these cases. The association of lesion presentation types with their site distribution revealed interesting trends. Plaques, the most common presentation, were predominantly seen on the upper limbs (56.5%), while papules, which made up 55.4% of the lesions, were most frequently observed on the upper limbs (66.7%). This finding aligns with the general understanding that certain skin lesions, such as papules, may prefer specific body areas like the upper limbs. The distribution of macules and patches was more evenly spread across the body, with macules appearing on both the upper and lower limbs, and patches being seen exclusively on the upper limbs. These findings provide valuable insights into the typical presentation patterns of pigmented lesions, which can aid in diagnosis. We did not find any relevant studies which relates to Histopathological Diagnosis and Association of presentation type & site distribution.

Comparison of concordance of clinical findings with histopathological findings and its comparison with other studies.

Correlation	Present study	Shushan S et al	Rajendra prasad Jagannadham et al[83]
Concordance with clinical finding	96.9%	78.9%	85%
In-concordance with clinical diagnosis	3.07%	21.1%	15%

This concordance with clinical diagnosis signifies the importance of Histopathological examination and sets it as gold standard for diagnosis of pigmented skin lesion.

Conclusion

CONCLUSION

- The study had 65 participants, with the majority falling in the 21-30 age group (32.3%) and a mean age of 33.5 years.
- A slightly higher proportion of male participants (52.3%) compared to females (47.7%) were observed.
- Lichen planus was the most common diagnosis, comprising 50.7% of the cases, followed by LP pigmentosus (13.8%).
- Itching was the most prevalent clinical feature, present in 98.5% of cases, while ulceration occurred in 30.8% of cases.
- Histopathological findings such as hyperkeratosis, acanthosis, and lymphocytic infiltrate were commonly observed and showed significant associations.
- The majority of lesions had a duration of more than one year (36.9%), followed by lifelong chronic lesions (32.3%).
- Most lesions were located on the upper limbs (63.1%), with plaques (35.4%) and papules (55.4%) being the most common presentation types.
- Special staining was rarely used, with most cases not requiring any special stains (72.3%).
- Concordance between clinical and histopathological diagnoses was generally high, with minor discrepancies observed in a few cases, such as hypertrophic LP and prurigo nodularis.
- The distribution of lesion types varied by body site, with plaques most frequently occurring on the upper limbs and papules being the most common overall presentation.
- Histopathological examination is gold standard for diagnosis of pigmented skin lesion.

Limitations

LIMITATIONS

- The study is limited by its hospital-based design, which may not be representative of the broader population, potentially limiting the generalizability of the findings.
- The cross-sectional nature of the study only provides a snapshot of data at a specific point in time, making it difficult to assess causal relationships or trends over time.
- The sample size of 65 participants may be insufficient to detect less common or subtle correlations between clinical and histopathological features of pigmented skin lesions.
- The study is limited to participants attending the Dermatology Department of Santosh Medical College and Hospital, which may introduce a selection bias, as the sample may not be diverse enough to reflect the general population's variety of pigmented lesions.
- The exclusion of individuals with hypopigmented skin lesions may limit the comprehensiveness of the study and exclude potentially relevant comparisons between different types of skin lesions.
- The study only includes participants aged 18 to 65, which may not fully capture the spectrum of pigmented skin lesions seen in younger or older age groups.
- Reliance on clinical and histopathological correlation may be limited by the expertise and accuracy of the pathologists and clinicians involved in the study.
- Participants' willingness to provide a biopsy sample is required, which may introduce a voluntary bias, as those willing to participate might have different characteristics than those who are not.

Summary

SUMMARY

The study entitled " **Clinico-Histopathological Study Of Pigmented Skin Lesions** " aims to establish a robust relationship between clinical and histopathological features of pigmented skin lesions. By examining 65 participants from Santosh Medical College and Hospital, the research aims to offer new insights into diagnosing and managing hyperpigmented lesions. Participants from the dermatology department, aged between 18 and 65, are chosen to provide a representative sample of the general population. Through the cross-sectional, observational design, the study correlates the clinical presentation of skin lesions with their histopathological findings, aiming to offer a more reliable approach to treatment.

Histology has long been recognized as an essential tool in diagnosing skin lesions, particularly in cases where clinical presentation alone is insufficient for definitive diagnosis. Pigmented lesions, such as those seen in conditions like melasma, post-inflammatory hyperpigmentation, and lichen planus pigmentosus, are common but often challenging to diagnose correctly. These lesions present with overlapping clinical characteristics, necessitating careful histopathological evaluation for a definitive diagnosis. The research, therefore, places a strong emphasis on histopathological examination as an adjunct to clinical assessment.

The results of this study are anticipated to offer significant value by facilitating early and accurate diagnosis, which, in turn, can lead to more effective management. In the study, the histological examination revealed crucial findings such as increased melanin deposition and alterations in keratinocyte morphology, especially in conditions like melasma. Furthermore, by documenting the clinical symptoms and the accompanying histopathological changes, such as lymphocytic infiltration in lichen planus pigmentosus, the study aims to refine the diagnostic accuracy for such conditions. The research also hopes to highlight the potential

of certain histopathological features as predictors of disease progression or response to treatment.

The study, however, faces limitations inherent to its design. First, as a hospital-based, cross-sectional study, the findings may not be generalizable to the wider population due to the specific patient demographics of the hospital. Additionally, the study's sample size of 65 participants may not be large enough to detect rarer conditions or subtle clinical-histopathological correlations. Furthermore, exclusion criteria based on the type of lesion and inadequate biopsy samples could introduce biases, limiting the scope of the findings. Another limitation arises from the study's reliance on the expertise of dermatopathologists and clinicians, as variations in diagnostic skills could influence the accuracy of the clinical-histopathological correlation.

Despite these limitations, the study's findings are expected to provide valuable contributions to the field of dermatology, particularly in improving the diagnostic pathways for pigmented skin lesions. By integrating both clinical and histopathological findings, it is hoped that the research will serve as a reference for future studies, further optimizing diagnostic practices in dermatology.

In conclusion, the research on histopathological and clinical correlation of hyperpigmented skin lesions is a significant endeavor in dermatology, offering critical insights that could potentially influence the future of skin lesion diagnosis and management. By combining clinical expertise with detailed histopathological analysis, the study aims to establish a clearer, more accurate understanding of the mechanisms behind skin pigmentation disorders, leading to more effective and personalized treatment approaches for patients with hyperpigmented skin lesions.

References

REFERENCES

1. Sarkar R, Puri P, Jain RK, Singh A, Desai A. Melasma in men: a clinical, aetiological and histological study. J Eur Acad Dermatol Venereal. 2010;24:768-72.

2. Khanna N, Rasool S. Facial melanoses: Indian Perspective. Indian J Dermatol Venereol Leprol. 2011;77:552-63.

3. Maize JC, Ackerman AB. Freckle. In: Maize JC, Ackerman AB, editors. Pigmented lesions of the skin. Philadelphia: Lea and Febiger; 1987:28-30.

4. Patel A, et al. Clinicopathological correlation of acquired hyperpigmentary disorders. J Dermatol. 2013;40(8):667-674. doi:10.1111/1346-8138.12236.

5. Rajput R, et al. Clinico-pathological study of non-neoplastic skin disorders: Correlation between clinical diagnosis and histopathological examination. Indian J Dermatol. 2017;62(5):552-558. doi:10.4103/0019-5154.211507.

6. Walker U, et al. Histopathological features in morphea (localized scleroderma) and their clinical correlates. J Am Acad Dermatol. 2017;77(6):1231-1238. doi:10.1016/j.jaad.2017.06.014.

7. Samanta A, et al. Histopathological study of skin tumors: A diagnostic challenge. Indian J Dermatol. 2018;63(6):498-503. doi:10.4103/ijd.ijd_211_18.

8. Dahlstrand A, et al. Extended-wavelength diffuse reflectance spectroscopy for tumor margin delineation in pigmented skin lesions. J Invest Dermatol. 2018;138(7):1606-1611. doi:10.1016/j.jid.2018.02.012.

9. Goswami R, Sharma V. Herbal extracts for treating skin hyperpigmentation disorders: A review. J Cosmet Dermatol. 2020;19(1):10-15. doi:10.1111/jocd.13112.

10. Day T, et al. Clinicopathologic diagnostic criteria for vulvar lichen planus. J Am Acad Dermatol. 2020;83(3):771-775. doi:10.1016/j.jaad.2020.03.043.

11. Ravindran D, et al. Histopathological and clinical correlations of hyperpigmented skin lesions. Indian J Dermatol. 2020;65(5):381-387. doi:10.4103/ijd.ijd_613_19.

12. Gangadhar A, et al. Correlations between clinical, dermoscopic, and histopathological findings in follicular keratotic diseases. Dermatol Pract Concept. 2021;11(1):e2021021. doi:10.5826/dpc.1101a21.

13. Farzinnia M, et al. Correlation between clinical and histopathological diagnoses in oral cavity lesions: A retrospective study. J Oral Pathol Med. 2022;51(7):585-590. doi:10.1111/jop.13215.

14. Kita R., Fraser H.B. Local adaptation of sun-exposure-dependent gene expression regulation in human skin. PLoS Genet. 2016;12:e1006382. doi: 10.1371/journal.pgen.1006382. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

15. Armenta A.M., Henkel E.D., Ahmed A.M. Pigmentation disorders in the elderly. Drugs Aging. 2019;36:235–245. doi: 10.1007/s40266-018-00633-w. [PubMed] [CrossRef] [Google Scholar]

16. Adigun C.G. Adverse drug reactions of the lower extremities. Clin. Podiatr. Med. Surg. 2016;33:397–408. doi: 10.1016/j.cpm.2016.02.007. [PubMed] [CrossRef] [Google Scholar]

17. Nicolaidou E., Katsambas A.D. Pigmentation disorders: Hyperpigmentation and hypopigmentation. Clin. Dermatol. 2014;32:66–72. doi: 10.1016/j.clindermatol.2013.05.026. [PubMed] [CrossRef] [Google Scholar]

18. Böhm M. Braun-Falco's Dermatology. Springer; Berlin/Heidelberg, Germany: 2021. Disorders of Melanin Pigmentation; pp. 1–35. [Google Scholar]

19. Ma E.Z., Zhou A.E., Hoegler K.M., Khachemoune A. Oculocutaneous albinism: Epidemiology, genetics, skin manifestation, and psychosocial issues. Arch. Dermatol. Res. 2023;315:107–116. doi: 10.1007/s00403-022-02335-1. [PubMed] [CrossRef] [Google Scholar]

20. Federico J.R., Krishnamurthy K. StatPearls. StatPearls Publishing; Treasure Island, FL, USA: 2022. Albinism. [PubMed] [Google Scholar]

21. Lee D.Y., Kim C.R., Lee J.H. Trichrome vitiligo in segmental type. Photodermatol. Photoimmunol. Photomed. 2011;27:111–112. doi: 10.1111/j.1600-0781.2011.00572.x. [PubMed] [CrossRef] [Google Scholar]

22. Hill J.P., Batchelor J.M. An approach to hypopigmentation. BMJ. 2017;356:356–362. doi: 10.1136/bmj.i6534. [PubMed] [CrossRef] [Google Scholar]

23. Silpa-Archa N., Kohli I., Chaowattanapanit S., Lim H.W., Hamzavi I. Postinflammatory hyperpigmentation: A comprehensive overview: Epidemiology, pathogenesis, clinical presentation, and noninvasive assessment technique. J. Am. Acad. Dermatol. 2017;77:591–605. doi: 10.1016/j.jaad.2017.01.035. [PubMed] [CrossRef] [Google Scholar]

24. Kallini J.R., Riaz F., Khachemoune A. Tinea versicolor in dark-skinned individuals. Int. J. Dermatol. 2014;53:137–141. doi: 10.1111/ijd.12345. [PubMed] [CrossRef] [Google Scholar]

25. Miazek N., Michalek I., Pawlowska-Kisiel M., Olszewska M., Rudnicka L. Pityriasis Alba—Common Disease, Enigmatic Entity: Up-to-Date Review of the Literature. Pediatr. Dermatol. 2015;32:786–791. doi: 10.1111/pde.12683. [PubMed] [CrossRef] [Google Scholar]

26. Jing J., Man X.Y. Vitiligo-like depigmentation in a patient treated with PD-1 antibody. BMJ. 2021;374:n1982. doi: 10.1136/bmj.n1982. [CrossRef] [Google Scholar]

27. McMichael L. Skin camouflage. BMJ. 2012;344:d7921. doi: 10.1136/bmj.d7921. [PubMed] [CrossRef] [Google Scholar]

28. Sheth P.B., Shah H.A., Dave J.N. Periorbital hyperpigmentation: A study of its prevalence, common causative factors and its association with personal habits and other disorders. Indian J. Dermatol. 2014;59:151–157. doi: 10.4103/0019-5154.127675. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

29. Choi W., Yin L., Smuda C., Batzer J., Hearing V.J., Kolbe L. Molecular and histological characterization of age spots. Exp. Dermatol. 2017;26:242–248. doi: 10.1111/exd.13203. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

30. Baxter L.L., Pavan W.J. The etiology and molecular genetics of human pigmentation disorders. Wiley Interdiscip. Rev. Dev. Biol. 2013;2:379–392. doi: 10.1002/wdev.72. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

31. Yadalla H.K.K., Aradhya S. Post acne hyperpigmentation: A brief review. Our Dermatol. Online. 2011;2:230–231. [Google Scholar]

32. Plensdorf S., Livieratos M., Dada N. Pigmentation disorders: Diagnosis and management. Am. Fam. Physician. 2017;96:797–804. [PubMed] [Google Scholar]

33. Woolery-Lloyd H., Kammer J.N. Seminars in Cutaneous Medicine and Surgery. Volume 30. WB Saunders; Philadelphia, PA, USA: 2011. Treatment of Hyperpigmentation; pp. 171–175. [PubMed] [Google Scholar]

34. Bala H.R., Lee S., Wong C., Pandya A.G., Rodrigues M. Oral tranexamic acid for the treatment of melasma: A review. Dermatol. Surg. 2018;44:814–825. doi: 10.1097/DSS.0000000000001518. [PubMed] [CrossRef] [Google Scholar]

35. Ali A.A., Al-Obaidi Z.M.J., Raauf A.M., Mahmood H.S. A Comparative, Randomized, Double-Blinded, and Vehicle-Controlled Study for the Reduction in Facial Pigmentation after Treatment with both Tranexamic Acid and Tranexamic Acid Ethyl Ester. Syst. Rev. Pharm. 2020;11:563–567. [Google Scholar]

36. Kaur A., Bhalla M., Sarkar R. Tranexamic acid in melasma: A review. Pigment Int. 2020;7:12–25. [Google Scholar]

37. Maeda K. Mechanism of Action of Topical Tranexamic Acid in the Treatment of Melasma and Sun-Induced Skin Hyperpigmentation. Cosmetics. 2022;9:108. doi: 10.3390/cosmetics9050108. [CrossRef] [Google Scholar]

38. McKesey J., Tovar-Garza A., Pandya A.G. Melasma treatment: An evidence-based review. Am. J. Clin. Dermatol. 2020;21:173–225. doi: 10.1007/s40257-019-00488-w. [PubMed] [CrossRef] [Google Scholar]

39. Grimes P.E., Ijaz S., Nashawati R., Kwak D. New oral and topical approaches for the treatment of melasma. Int. J. Women's Dermatol. 2019;5:30–36. doi: 10.1016/j.ijwd.2018.09.004. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

40. Artzi O., Horovitz T., Bar-Ilan E., Shehadeh W., Koren A., Zusmanovitch L., Mehrabi J., Salameh F., Nelkenbaum G.I., Zur E., et al. The pathogenesis of melasma and implications for treatment. J. Cosmet. Dermatol. 2021;20:3432–3445. doi: 10.1111/jocd.14382. [PubMed] [CrossRef] [Google Scholar]

41. Taraz M., Niknam S., Ehsani A.H. Tranexamic acid in treatment of melasma: A comprehensive review of clinical studies. Dermatol. Ther. 2017;30:e12465. doi: 10.1111/dth.12465. [PubMed] [CrossRef] [Google Scholar]

42. Tse T.W., Hui E. Tranexamic acid: An important adjuvant in the treatment of melasma. J. Cosmet. Dermatol. 2013;12:57–66. doi: 10.1111/jocd.12026. [PubMed] [CrossRef] [Google Scholar]

43. Sharma R., Mahajan V.K., Mehta K.S., Chauhan P.S., Rawat R., Shiny T.N. Therapeutic efficacy and safety of oral tranexamic acid and that of tranexamic acid local infiltration with microinjections in patients with melasma: A comparative study. Clin. Exp. Dermatol. 2017;42:728–734. doi: 10.1111/ced.13164. [PubMed] [CrossRef] [Google Scholar]

44. Kim S.J., Park J.Y., Shibata T., Fujiwara R., Kang H.Y. Efficacy and possible mechanisms of topical tranexamic acid in melasma. Clin. Exp.

Dermatol. 2016;41:480–485. doi: 10.1111/ced.12835. [PubMed] [CrossRef] [Google Scholar]

45. Sofen B., Prado G., Emer J. Melasma and post inflammatory hyperpigmentation: Management update and expert opinion. Skin. Ther. Lett. 2016;21:1–7. [PubMed] [Google Scholar]

46. Demir B., Çiçek D., Bilik L., Aydoğdu E.G., Artaş H., Demirpolat N., Ergin C. Oral isotretinoin induced pigmentation disorder: A case report. Firat Tip Derg. 2017;22:143–145. [Google Scholar]

47. Mysore V., Mahadevappa O.H., Barua S., Majid I., Viswanath V., Bhat R.M., Talwar S., Thurakkal S., Aurangabadkar S.J., Chatterjee M., et al. Standard guidelines of care: Performing procedures in patients on or recently administered with isotretinoin. J. Cutan. Aesthetic Surg. 2017;10:186–194. doi: 10.4103/JCAS.JCAS_110_17. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

48. Bagatin E., Costa C.S. The use of isotretinoin for acne–an update on optimal dosing, surveillance, and adverse effects. Expert Rev. Clin. Pharmacol. 2020;13:885–897. doi: 10.1080/17512433.2020.1796637. [PubMed] [CrossRef] [Google Scholar]

49. Fallah H., Rademaker M. Isotretinoin in the management of acne vulgaris: Practical prescribing. Int. J. Dermatol. 2021;60:451–460. doi: 10.1111/ijd.15089. [PubMed] [CrossRef] [Google Scholar]

50. Villani A., Nastro F., Di Vico F., Fabbrocini G., Annunziata M.C., Genco L. Oral isotretinoin for acne: A complete overview. Expert Opin. Drug Saf. 2022;21:1027–1037. doi: 10.1080/14740338.2022.2102605. [PubMed] [CrossRef] [Google Scholar]

51. Spring L.K., Krakowski A.C., Alam M., Bhatia A., Brauer J., Cohen J., Rosso J.Q., Diaz L., Dover J., Eichenfield L.F., et al. Isotretinoin and timing of procedural interventions: A systematic review with consensus recommendations. JAMA Dermatol. 2017;153:802–809. doi: 10.1001/jamadermatol.2017.2077. [PubMed] [CrossRef] [Google Scholar]

52. Chu S., Michelle L., Ekelem C., Sung C.T., Rojek N., Mesinkovska N.A. Oral isotretinoin for the treatment of dermatologic conditions other than acne: A systematic review and discussion of future directions. Arch. Dermatol. Res. 2021;313:391–430. doi: 10.1007/s00403-020-02152-4. [PubMed] [CrossRef] [Google Scholar]

53. Shao X., Chen Y., Zhang L., Zhang Y., Ariyawati A., Chen T., Chen J., Liu L., Pu Y., Li Y., et al. Effect of 30% supramolecular salicylic acid peel on skin microbiota and inflammation in patients with moderate-to-severe acne vulgaris. Dermatol. Ther. 2023;13:155–168. doi: 10.1007/s13555-022-00844-5. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

54. Dréno B., Araviiskaia E., Kerob D., Andriessen A., Anfilova M., Arenbergerova M., Barrios L.O., Mokos Z.B., Haedersdal M., Hofmann M.A. Nonprescription acne vulgaris treatments: Their role in our treatment armamentarium—An international panel discussion. J. Cosmet. Dermatol. 2020;19:2201–2211. doi: 10.1111/jocd.13497. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

55. Rachmin I., Ostrowski S.M., Weng Q.Y., Fisher D.E. Topical treatment strategies to manipulate human skin pigmentation. Adv. Drug Deliv. Rev. 2020;153:65–71. doi: 10.1016/j.addr.2020.02.002. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

56. Bose S.K., Ortonne J.P. Textbook of Cosmetic Dermatology. Martin-Dunitz Ltd.; London, UK: 1998. Pigmentation: Dyschromia; pp. 391–415. [Google Scholar]

57. Yasir M., Goyal A., Sonthalia S. StatPearls. StatPearls Publishing; Treasure Island, FL, USA: 2022. Corticosteroid adverse effects. [PubMed] [Google Scholar]

58. Yélamos O., Alejo B., Ertekin SSVilla-Crespo L., Zamora-Barquero S., Martinez N., Domínguez M., Iglesias P., Herrero A., Malvehy J., Puig S. Non-invasive clinical and microscopic evaluation of the response to treatment with clobetasol cream vs. calcipotriol/betamethasone dipropionate foam in mild to moderate plaque psoriasis: An investigator-initiated, phase IV, unicentric, open, randomized clinical trial. J. Eur. Acad. Dermatol. Venereol. 2021;35:143–149. [PMC free article] [PubMed] [Google Scholar]

59. Cho J.H., Bhutani S., Kim C.H., Irwin M.R. Anti-inflammatory effects of melatonin: A systematic review and meta-analysis of clinical trials. Brain Behav. Immun. 2021;93:245–253. doi: 10.1016/j.bbi.2021.01.034. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

60. Karekar S.R., Marathe P.A., Nagarajan V.B., Khopkar U.S., Chikhalkar S.B., Desai P.K., Dongre M.S. Use of topical steroids in dermatology: A questionnaire based study. Indian Dermatol. Online J. 2020;11:725–730. [PMC free article] [PubMed] [Google Scholar]

61. Chadderdon C., Gaston R.G., Loeffler B.J., Lewis D. Betamethasone Versus Ketorolac Injection for the Treatment of De Quervain's Tenosynovitis: A Double-Blind Randomized Clinical Trial: Level 1 Evidence. J. Hand Surg. 2017;42:S45–S46. doi: 10.1016/j.jhsa.2017.06.086. [CrossRef] [Google Scholar]

62. Patel H.K., Barot B.S., Parejiya P.B., Shelat P.K., Shukla A. Topical delivery of clobetasol propionate loaded microemulsion based gel for effective treatment of vitiligo: Ex vivo permeation and skin irritation studies. Colloids Surf. B Biointerfaces. 2013;102:86–94. doi: 10.1016/j.colsurfb.2012.08.011. [PubMed] [CrossRef] [Google Scholar]

63. Khaitan B.K., Sindhuja T. Autoimmunity in vitiligo: Therapeutic implications and opportunities. Autoimmun. Rev. 2022;21:102932. doi: 10.1016/j.autrev.2021.102932. [PubMed] [CrossRef] [Google Scholar]

64. Eleftheriadou V., Atkar R., Batchelor J., McDonald B., Novakovic L., Patel J.V., Ravenscroft J., Rush E., Shah D., Shah R., et al. British Association of Dermatologists' Clinical Standards Unit. British Association of Dermatologists guidelines for the management of people with vitiligo 2021. Br. J. Dermatol. 2022;186:18–29. doi: 10.1111/bjd.20596. [PubMed] [CrossRef] [Google Scholar]

65. Habet K.A., Kolli S.S., Pona A., Feldman S.R. A review of topical corticosteroid sprays for the treatment of inflammatory dermatoses. Dermatol. Online J. 2019;25:3–12. doi: 10.5070/D3258045123. [PubMed] [CrossRef] [Google Scholar]

66. Gajinov Z. Corticosteroid topical therapy range: Fluocinolone-acetonide gel. Galen. Med. J. 2022;1:17–22. doi: 10.5937/Galmed2202017G. [CrossRef] [Google Scholar]

67. Medici S., Peana M., Nurchi V.M., Zoroddu M.A. Medical uses of silver: History, myths, and scientific evidence. J. Med. Chem. 2019;62:5923–5943. doi: 10.1021/acs.jmedchem.8b01439. [PubMed] [CrossRef] [Google Scholar]

68. Bandyopadhyay D. Topical antibacterials in dermatology. Indian J. Dermatol. 2021;66:117–125. doi: 10.4103/ijd.IJD_99_18. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

69. Adhya A., Bain J., Ray O., Hazra A., Adhikari S., Dutta G., Ray S., Majumdar B.K. Healing of burn wounds by topical treatment: A randomized controlled comparison between silver sulfadiazine and nano-crystalline silver. J. Basic Clin. Pharm. 2014;6:29–34. doi: 10.4103/0976-0105.145776. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

70. Nethi S.K., Das S., Patra C.R., Mukherjee S. Recent advances in inorganic nanomaterials for wound-healing applications. Biomater. Sci. 2019;7:2652–2674. doi: 10.1039/C9BM00423H. [PubMed] [CrossRef] [Google Scholar]

71. Srivastava S., Patil A., Prakash C., Kumari H. Comparison of intralesional triamcinolone acetonide, 5-fluorouracil, and their combination in treatment of keloids. World J. Plast. Surg. 2018;7:212–219. doi: 10.1089/wound.2017.0741. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

72. Khan H.A., Sahibzada M.N., Paracha M.M. Comparison of the efficacy of intralesional bleomycin versus intralesional triamcinolone acetonide in the treatment of keloids. Dermatol. Ther. 2019;32:e13036-40. doi: 10.1111/dth.13036. [PubMed] [CrossRef] [Google Scholar]

73. Raman P., Pitty R., Krithika C.L., Anand S.N., Subramani G.P. Topical curcumin and triamcinolone acetonide in recurrent minor aphthous ulcers: A pilot trial. J. Contemp. Dent. Pract. 2020;21:884–890. [PubMed] [Google Scholar]

74. Kwiatkowska D., Wicka M., Bulska E., Kaliszewski P. Investigation of the Excretion of Triamcinolone Acetonide and Its Metabolite. Separations.

2023;10:164. doi: 10.3390/separations10030164. [CrossRef] [Google Scholar]

75. Mangion S.E., Mackenzie L., Roberts M.S., Holmes A.M. Seborrheic dermatitis: Topical therapeutics and formulation design. Eur. J. Pharm. Biopharm. 2023;185:148–164. doi: 10.1016/j.ejpb.2023.01.023. [PubMed] [CrossRef] [Google Scholar]

76. Pinto L.M., Chiricozzi A., Calabrese L., Mannino M., Peris K. Novel Therapeutic Strategies in the Topical Treatment of Atopic Dermatitis. Pharmaceutics. 2022;14:2767. doi: 10.3390/pharmaceutics14122767. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

77. Sandhu S., Klein B.A., Al-Hadlaq M., Chirravur P., Bajonaid A., Xu Y., Intini R., Hussein M., Vacharotayangul P., Sroussi H., et al. Oral lichen planus: Comparative efficacy and treatment costs—A systematic review. BMC Oral Health. 2022;22:161. doi: 10.1186/s12903-022-02168-4. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

78. Pakravan F., Ghalayani P., Emami H., Isfahani M.N., Noorshargh P. A novel formulation for radiotherapy-induced oral mucositis: Triamcinolone acetonide mucoadhesive film. J. Res. Med. Sci. Off. J. Isfahan Univ. Med. Sci. 2019;24:63. [PMC free article] [PubMed] [Google Scholar]

79. Mangold A.R., Pittelkow M.R. Lichen planus. Clin. Basic Immunodermatology. 2017:551–576. doi: 10.1007/978-3-319-29785-9_31. [CrossRef] [Google Scholar]

80. Ravindran S, Teja KR, Kumar SP, Arnold J, Balaji S. Histopathological and clinical correlation of hyperpigmented skin lesions. Sch J App Med Sci. 2020;8(11):2472-7. doi:10.36347/sjams.2020.v08i11.007.

81. Mruthyunjayappa S, Hemalata M, Gopal MG, Suguna BV. A study of spectrum of histopathological features in patients presenting with hyperpigmented skin lesions. Arch Med Health Sci. 2016;4(2):189. doi:10.4103/2321-4848.196195.

82. Jaiswal A, Avani, Gupta K, Jain M, et al. Histopathological spectrum of hyperpigmented skin lesions – A study in a tertiary hospital from western Uttar Pradesh. Panacea J Med Sci. 2023;13(1):155-9. doi:10.18231/j.pjms.2023.032.

83. Rajendra Prasad J, Suryakala C, Arfathunnisa M, et al. Clinico pathological study of pigmented skin lesions. J. Evid. Based Med. Healthc. 2020; 7(7), 337-341. doi: 10.18410/jebmh/2020/72.

84. Jayker, Shushan Shweta et al. "HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED LESIONS OF SKIN." Journal of Evolution of medical and Dental Sciences 5 (2016): 1913-1916.

Master Chart

MASTER CHART

S.No.	UHID	AGE	Age	GENDER	DIAGNOSIS	Itching	Ulceration	Hyperkeratosis	Parakeratosis	Acanthosis	Band_Like_Infiltrate	Pigment_Incontinence	Hypergranulosis	Spongiosis	Lymphocytic_Infiltrate	Perivascular_Infiltration	Max_Joseph_Space	Saw_Toothed_Rete_Ridges	Duration_of_Lesion	Site_Distribution	Presentation_Type	Special_Stains	Clinical_Findings	Histopathological_Findings
1	202404271305	33	31-40	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
2	202405010751	33	31-40	M	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	Present	Present	Lifelong	Upper limbs	Papules	Crystal Violet	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
3	202404290381	62	>60	F	LP chronicus	Present	Present	Present	Absent	Absent	Absent	Absent	Absent	Present	Present	Present	Absent	Absent	>1 year	Upper limbs	Plaques	None	Thickened plaques with chronic itching	Epidermal hyperplasia, Hypergranulosis, Chronic inflammation
4	202404300974	22	21-30	F	BECKERS NEVUS	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	Present	Present	Absent	Lifelong	Upper limbs	Macules	None	Hyperpigmented patch, sometimes with hair growth	Hyperpigmentation, Occasional smooth muscle proliferation, Acanthosis
5	202404231014	40	31-40	F	LP simplex	Present	Present	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Present	<6 months	Upper limbs	Plaques	None	Localized pruritic plaques, Chronic itching	Thickened epidermis, Minimal inflammation
6	2404250371	27	21-30	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Both upper and lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
7	202404080292	32	31-40	F	LP chronicus	Present	Present	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Present	>1 year	Whole body	Plaques	PAS	Thickened plaques with chronic itching	Epidermal hyperplasia, Hypergranulosis, Chronic inflammation
8	202401230962	19	≤20	F	LP pigmentosus	Present	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	>1 year	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
9	202310110506	22	21-30	F	Hypertrophic LP	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Absent	Present	Present	Absent	Present	>1 year	Whole body	Plaques	None	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate
10	202401020356	24	21-30	M	LP pigmentosus	Present	Present	Absent	Absent	Present	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Lifelong	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate

11	202312070516	25	21-30	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
12	202311090858	20	≤20	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
13	202302200698	22	21-30	F	Nevus sebaceous	Present	Absent	Present	Absent	Absent	Absent	Present	Absent	Absent	Present	Absent	Present	Absent	Lifelong	Whole body	Macules	None	Patch or Hairless area	Hyperkeratosis, sebaceous gland hyperplasia
14	202303150397	22	21-30	M	LP pilaris	Present	Absent	Present	Present	Absent	Absent	Present	Present	Absent	Absent	Absent	Absent	Present	<6 months	Upper limbs	Plaques	None	Follicular papules, itching	Follicular plugging, hyperkeratosis, lymphocytic infiltrate
15	202303140374	20	≤20	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Upper limbs	Papules	PAS	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
16	202303090482	27	21-30	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
17	17109324	31	31-40	M	LP pigmentosus	Present	Absent	Absent	Absent	Absent	Absent	Absent	Present	Absent	Present	Present	Absent	Present	>1 year	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
18	17202571	20	≤20	M	Hypertrophic LP	Present	Present	Present	Absent	Absent	Absent	Present	Absent	Absent	Absent	Present	Absent	Absent	Lifelong	Upper limbs	Plaques	PAS	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate
19	17197460	36	31-40	F	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
20	17196989	27	21-30	F	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
21	17192362	42	41-50	F	Nevus sebaceous	Present	Present	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	Present	Absent	Absent	>1 year	Upper limbs	Patches	None	Patch or Hairless area	Hyperkeratosis, sebaceous gland hyperplasia
22	17185353	28	21-30	M	LP pigmentosus	Present	Absent	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	Absent	Absent	Absent	>1 year	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate

23	202306020561	45	41-50	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Present	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
24	17253071	24	21-30	M	Nevus sebaceous	Present	Present	Absent	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	Absent	Absent	Lifelong	Whole body	Macules	Fontana Masson	Patch or Hairless area	Hyperkeratosis, sebaceous gland hyperplasia
25	202302080381	31	31-40	M	LP pigmentosus	Present	Present	Absent	Absent	Absent	Absent	Present	Absent	Absent	Present	Absent	Present	Absent	<6 months	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
26	202302040311	22	21-30	M	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
27	202309010626	42	41-50	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Present	Present	Present	Lifelong	Both upper and lower limbs	Papules	Crystal Violet	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
28	202308120721	40	31-40	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
29	202302030091	65	>60	F	Basal cell carcinoma	Present	Present	Absent	Absent	Present	Absent	Absent	Absent	Present	Present	Present	Absent	Present	<6 months	Whole body	Plaques	None	Other clinical findings	Other histopathological findings
30	202302200698	26	21-30	M	Nevus sebaceous	Present	Present	Absent	Absent	Present	Absent	Absent	Present	Present	Present	Absent	Absent	Absent	Lifelong	Upper limbs	Patches	None	Patch or Hairless area	Hyperkeratosis, sebaceous gland hyperplasia
31	17245013	52	51-60	M	LP pigmentosus	Present	Present	Present	Absent	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	<6 months	Whole body	Plaques	Fontana Masson	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
32	17200141	42	41-50	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
33	17232350	18	≤20	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
34	17229448	20	≤20	F	LP pigmentosus	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	>1 year	Upper limbs	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate

35	202305130377	43	41-50	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
36	202305090436	40	31-40	M	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
37	202303150397	22	21-30	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Present	Present	Present	Present	Present	<6 months	Both upper and lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
38	202303140374	20	≤20	M	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Upper limbs	Papules	Crystal Violet	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
39	20230309048	33	31-40	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	<6 months	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
40	202302270677	32	31-40	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
41	17175376	18	≤20	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	Crystal Violet	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
42	17180874	45	41-50	F	Lichen planus	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Upper limbs	Papules	Fontana Masson	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
43	17138573	31	31-40	M	LP pigmentosus	Present	Present	Absent	Absent	Absent	Absent	Present	Present	Present	Present	Absent	Absent	Absent	>1 year	Whole body	Plaques	None	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
44	171422521	21	21-30	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	Lifelong	Both upper and lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
45	17140751	35	31-40	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space

46	17140992	31	31-40	F	LP pigmentosus	Present	Absent	Present	Present	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Absent	<6 months	Whole body	Plaques	Fontana Masson	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
47	17139121	21	21-30	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	Fontana Masson	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
48	17111576	32	31-40	M	Hypertrophic LP	Present	Present	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	Absent	Present	Present	<6 months	Upper limbs	Plaques	None	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate
49	17122875	29	21-30	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	<6 months	Upper limbs	Papules	PAS	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
50	17122914	52	51-60	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	Lifelong	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
51	17210128	37	31-40	M	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration, Max Joseph Space
52	17210341	56	51-60	M	Hypertrophic LP	Present	Present	Absent	Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	Present	Present	Lifelong	Upper limbs	Plaques	None	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate
53	17076672	27	21-30	M	LP pigmentosus	Present	Present	Absent	Absent	Present	Absent	Present	Absent	Present	Absent	Absent	Absent	Absent	Lifelong	Whole body	Plaques	Crystal Violet	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
54	17103687	45	41-50	F	Hypertrophic LP	Present	Present	Absent	Absent	Present	Absent	Present	Absent	Present	Absent	Absent	Present	Absent	>1 year	Whole body	Plaques	None	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis, epidermal hyperplasia, chronic inflammatory infiltrate
55	17102909	30	21-30	F	Lichen planus	Present	Absent	Present	Absent	Present	Present	Present	Present	Absent	Present	Absent	Present	Present	>1 year	Upper limbs	Papules	PAS	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration
56	174654647	39	31-40	F	Prurigo Nodularis	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Present	Absent	Absent	Present	<6 months	Both upper and lower limbs	Plaques	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Band-like lymphocytic infiltration,Hypergranulosis, Orthokeratosis,Pseudoepitheliomatous hyperplasia,Dermal fibrosis

57	20231025069	53	51-60	F	Prurigo Simplex	Present	Absent	Present	Absent	Present	Present	Absent	Absent	Present	Present	Present	Absent	Absent	<6 months	Lower limbs	Papules	None	Thickened hyperkeratotic plaques, chronic itching	Acanthosis, Hyperkeratosis, Band-like inflammatory infiltrate
58	202410056784	54	51-60	M	Prurigo Nodularis	Present	Absent	Present	Present	Present	Absent	Present	Present	Absent	Present	Absent	Absent	Absent	>1 year	Upper limbs	Plaques	None	Violaceous papules and plaques, Itching	Hyperkeratosis,Parakeratosis, Band-like lymphocytic infiltration, Hypergranulosis
59	2023010280824	18	≤20	F	Prurigo Simplex	Present	Absent	Present	Absent	Absent	Present	Absent	Absent	Absent	Present	Present	Absent	Absent	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Band-like lymphocytic infiltration
60	17472600	34	31-40	F	Lichen Amylodosis	Absent	Absent	Present	Absent	Present	Present	Present	Absent	Absent	Present	Present	Absent	Absent	>1 year	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Acanthosis, Hyperkeratosis,Band-like inflammatory infiltrate, Pigmentary incontonence, Basal layer pigmentation
61	202404278931	73	>60	M	Prurigo Nodularis	Present	Absent	Absent	Present	Present	Absent	Present	Present	Absent	Present	Absent	Absent	Absent	<6 months	Upper limbs	Plaques	None	Violaceous papules and plaques, Itching	Parakeratosis,Acanthosis, Pseudoepitheliomatous hyperplasia,Dermal Fibrosis,Band like lymphocytic infiltrate, Hypergranulosis
62	202404271389	41	41-50	M	Discoid Lupus Erythematous	Present	Absent	Present	Absent	Present	Present	Present	Absent	Present	Present	Present	Absent	Absent	Lifelong	Lower limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis,Follicular keratotic plugging, Band-like lymphocytic infiltration,Pigmentary Incontinence
63	202410998765	63	>60	F	LP pigmentosus	Present	Present	Absent	Absent	Present	Absent	Absent	Absent	Absent	Present	Present	Absent	Present	Lifelong	Whole body	Plaques	Fontana Masson	Hyperpigmented macules/patches, mild itching	Basal layer hyperpigmentation, sparse lymphocytic infiltrate
64	17490097	69	>60	M	Seborrhic Keratosis	Present	Present	Present	Absent	Present	Absent	Absent	Present	Absent	Present	Present	Absent	Present	<6 months	Upper limbs	Plaques	None	Thickened hyperkeratotic plaques, chronic itching	Hyperkeratosis,Acanthosis, Papillomatosis Horn cyst filled with Keratin, epidermal hyperplasia, chronic inflammatory infiltrate
65	202404271841	32	31-40	F	Nevus sebaceous	Present	Absent	Present	Present	Present	Present	Present	Present	Absent	Present	Present	Present	Present	Lifelong	Upper limbs	Papules	None	Violaceous papules and plaques, Itching	Hyperkeratosis, Acanthosis, Band-like lymphocytic infiltration,Basal layer pigmentation

Annexures

ANNEXURES

CLNICAL PROFROMA

Name of the Patient:

Age:

Gender:

Occupation:

Chief Complaints and duration of the illness:

History of Present illness:

1. History of itching
2. History of pain
3. History of burning sensation
4. History of loss of sensation
5. History of excess sweating
6. History of photosensitivity

Past history:

Family history:

Treatment history:

General Examination:

Dermatological examination:

(I) Examination of the skin:

a) Number of hyperpigmented lesions

b) Site of the lesions

c) Size of the lesions

d) Well defined / ill defined

e) Scaling / non-scaling

f) Sensation – intact / diminished

g) Peripheral nerves enlargement

(II) Examination of the mucosa:

(III)Examination of the hair:

(IV) Examination of the nails:

CLINICAL DIAGNOSIS

CONSENT FORM (ENGLISH)

I_____ age_____ sex_____ give my full free and valid consent to be subject in the study titled " **Clinico-Histopathological Study Of Pigmented Skin Lesions** " I have been informed to my full satisfaction by Dr _____ about the purpose, nature and various laboratory investigations for the study to be carried out. I have been informed about the duration of study and instructions to be followed during study period. I realize that this is being done for the sake of knowing the changes in the glycated haemoglobin levels due to iron deficiency anaemia. This can prevent us from taking any wrong therapeutic decision based on glycated haemoglobin level without confirming iron deficiency anaemia status. I am aware of my right to opt out of study at any time during the course of study.

Date:

Patient/patient's relative's

Place:
Signature or Thumb Impression

Name:

Signature of treating physician

Date:

Place:

Name:

Signature of witness:

सहमति पत्र

दिनांक–

मैं ..... उम्र ..... निवासी .... घोषणा करता/करती हूँ कि मैं थीसिस अध्ययन में भाग लेने के लिए अपनी सहमति देता/देती हूँ , जिसका शीर्षक है '' तृतीयक देखभाल केंद्र में रंजित त्वचा के घावों का हिस्टोपैथोगिकल और नैदानिक सहसंबंध'' मैंनें रोगी सूचना पत्र और इसके बारे में सारे प्रश्नों को अच्छी तरह पढ़ लिया है। इस अध्ययन का डाटा इस शर्त पर है कि मेरे सभी विवरण गोपनीय रखे जाने हैं।

मैं संतोष अस्पताल, गाजियाबाद उ0प्र0 में अपने आगे के इलाज का का अधिकारी तथा जब भी मैं जाना चाहता/चाहती हूँ या अध्ययन से अपने हटने के अधिकार को सुरक्षित रखता हूँ।

मरीज के हस्ताक्षर	नाम
रिश्तेदार के हस्ताक्षर	नाम
गवाह के हस्ताक्षर	नाम
डाक्टर के हस्ताक्षर	नाम

तिथि –

ETHICAL CLEARANCE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/R/2023/2489[92] Dated: 26.09.2023

Subject: Grant of Ethical Clearance for **Thesis Protocol** Topic **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS",** submitted by **Dr. SWARNIM BAIDYA,** MD/MS student of Batch 2022-2023, Department of **PATHOLOGY**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"CLINICO-HISTOPATHOLOGICAL STUDY OF PIGMENTED SKIN LESIONS", Dr. SWARNIM BAIDYA** is informed that the **Thesis Protocol** submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 17.07.2023 to 28.07.2023. The recommendations of the **Screening Committee** were considered in detail by the **Institutional Ethics Committee** in its meeting held on **23.09.2023** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. NARESH SHARMA]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR DELHI

Dr. SWARNIM BAIDYA
MD/MS student of batch 2022-2023
Department of PATHOLOGY
Email: sbaidya028@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Academics
4. The Dean – Research
5. The Director - IQAC
6. Dr. Swati Singh (Guide)
7. HOD of PATHOLOGY

Dissertation Workshop Certificate

SANTOSH
Deemed to be University

Celebrating 30 Impactful years
Since 1990

Workshop on Dissertation Writing
16th February 2025

Certificate Of Participation

Is presented to Dr Swarnim Baidya

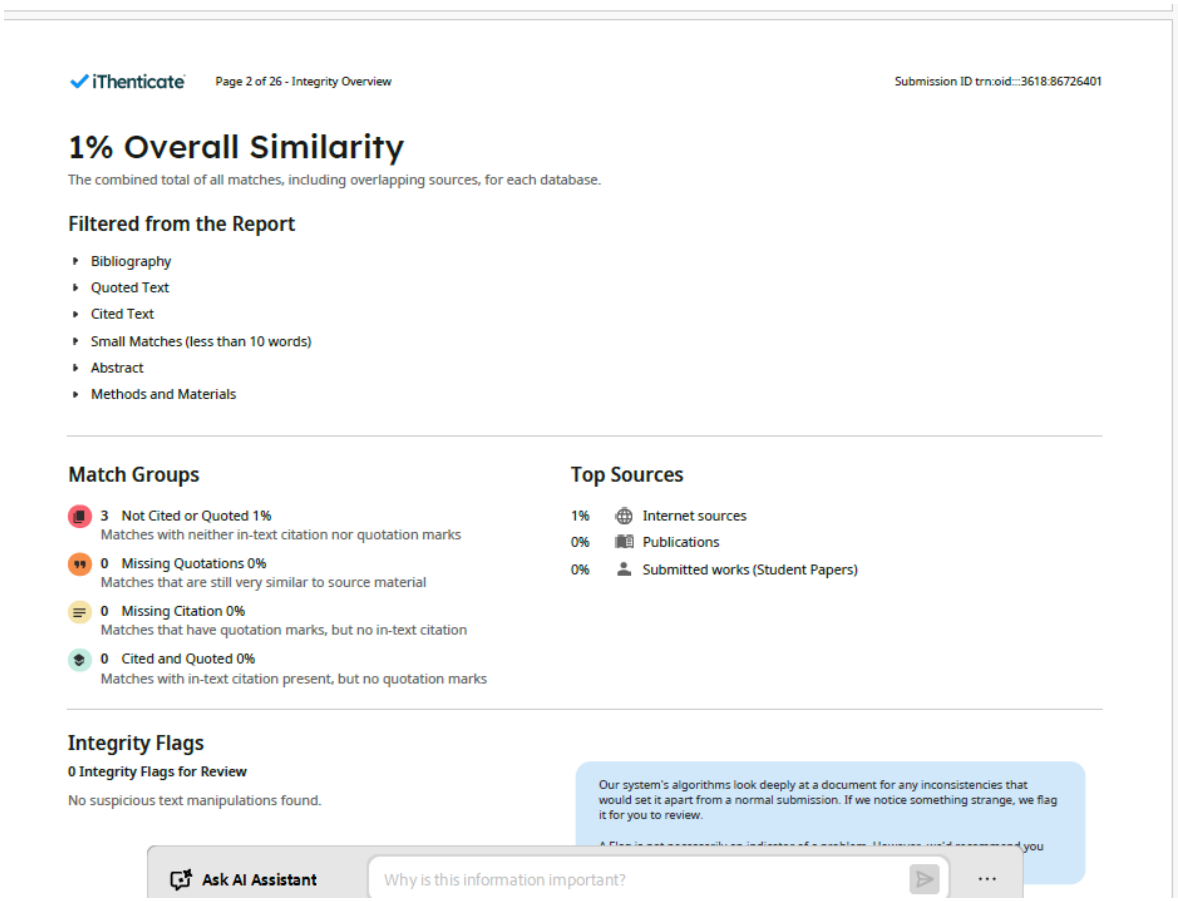
For his/her participation as a faculty/ delegate/ in
Workshop on Dissertation Writing

ORGANIZED BY
Santosh Medical College & Hospitals
Ghaziabad (Delhi NCR).

Dr Alka Agarwal
Dean and Professor, SMCH Ghaziabad

Mahalakshmi
Dr. Mahalakshmi, V.N
Vice Chancellor

Plagiarism certificate



iThenticate Page 2 of 26 - Integrity Overview Submission ID trn:oid:::3618:86726401

1% Overall Similarity

The combined total of all matches, including overlapping sources, for each database.

Filtered from the Report

- Bibliography
- Quoted Text
- Cited Text
- Small Matches (less than 10 words)
- Abstract
- Methods and Materials

Match Groups

3 Not Cited or Quoted 1%
Matches with neither in-text citation nor quotation marks

0 Missing Quotations 0%
Matches that are still very similar to source material

0 Missing Citation 0%
Matches that have quotation marks, but no in-text citation

0 Cited and Quoted 0%
Matches with in-text citation present, but no quotation marks

Top Sources

1% Internet sources
0% Publications
0% Submitted works (Student Papers)

Integrity Flags

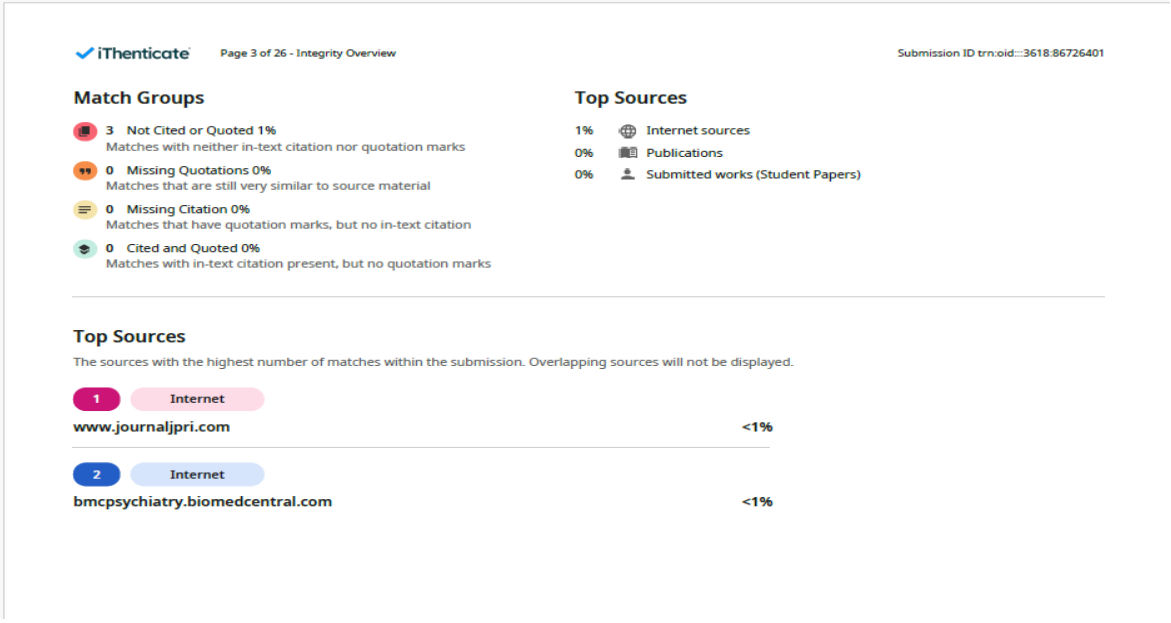
0 Integrity Flags for Review

No suspicious text manipulations found.

Our system's algorithms look deeply at a document for any inconsistencies that would set it apart from a normal submission. If we notice something strange, we flag it for you to review.

Ask AI Assistant Why is this information important?

iThenticate Page 2 of 26 - Integrity Overview Submission ID trn:oid:::3618:86726401



iThenticate Page 3 of 26 - Integrity Overview Submission ID trn:oid:::3618:86726401

Match Groups

3 Not Cited or Quoted 1%
Matches with neither in-text citation nor quotation marks

0 Missing Quotations 0%
Matches that are still very similar to source material

0 Missing Citation 0%
Matches that have quotation marks, but no in-text citation

0 Cited and Quoted 0%
Matches with in-text citation present, but no quotation marks

Top Sources

1% Internet sources
0% Publications
0% Submitted works (Student Papers)

Top Sources

The sources with the highest number of matches within the submission. Overlapping sources will not be displayed.

1 Internet
www.journaljpri.com <1%

2 Internet
bmcpsychiatry.biomedcentral.com <1%